Interaction Between Smoking and Body Weight: Implications for Public Health Policy

by

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DEDICATION

To my mother and father for their love and support.

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ABSTRACT

Cigarette consumption and excess weight are the two leading causes of premature death and disability in the US. Smoking cessation has well-documented health benefits. However, 80% of quitters experience an unintended consequence of quitting: post-cessation weight gain. Currently, the health message provided to smokers is that it is never too late to quit, without addressing the weight concern that many have.

I outline three papers in this dissertation to study the interaction of smoking behaviors and body weight. The first paper examines the age-specific changes in BMI and BMI distribution among US adults, adjusting for smoking and socioeconomic status (SES). I examine population representative annual BMI change across two time periods (1997 to 2007 and 2007 to 2017) by age, gender, SES and smoking status using a synthetic cohort approach. I also assess the changes in BMI distribution among age-specific groups. Information on BMI change and BMI distribution change can help us identify age-groups that are more susceptible to excess weight gain. The second study investigates the tradeoff between the health benefits of quitting smoking and the harm due to post-cessation weight gain. I estimate the break-even weight gain, i.e. the weight individuals would need to gain to offset the benefits of smoking cessation. A potentially attainable break-even weight gain exists for certain combinations of quit-age and BMI. The break-even weight gain decreases with quit-age. The break-even weight gain for white males with an initial BMI of 30 is 29.9 kg if they quit at 50 but decreases to 4.2 kg if they quit 80. I identify subgroups with higher initial BMI and higher cigarette consumption as high-risk to receive negative net health benefits from quitting. Smoking

cessation should be coupled with weight management programs to maximize health gains. Lastly, I explore interventions that could reduce post-cessation weight gain and estimate the potential increase of overall welfare in the population. Employing an agent-based model, I simulate three interventions on a nationally representative sample of US smokers: pharmacotherapy, physical activity intervention and alternative tobacco product (electronic cigarette). These interventions affect both smoking cessation rates and post-cessation weight gain. Model results show that electronic cigarette is the dominant intervention that increases life-years saved and reduces obesity prevalence.

INTRODUCTION

The two leading causes of premature death and disability in the US are cigarette consumption and excess weight [1]. It is well known that smoking can lead to many adverse health conditions such as cardiovascular diseases, lung cancer and exacerbation of multiple chronic health conditions [2]. Tobacco smoking has remained the leading preventable cause of disease and premature death in the US [3,4]. According to the Surgeon General's Report on smoking in 2014, the estimated annual smoking-attributable death is around 480,000 [4]. Policy-makers have implemented interventions to either prevent non-smokers from initiating or encourage smokers to quit. Common interventions include cigarette excise taxes, smoke-free air laws, nicotine replacement treatment and cognitive behavioral therapy [5-8].

Smoking cessation benefits not only individual smokers, but also the society as a whole. Studies have found that the all-cause mortality rate is about three times among smokers compared to those who have never smoked [9]. More specifically, the relative risk of mortality for smokers is around 25 for lung cancer, 2 from renal failure, 6 from intestinal ischemia, 2.4 from hypertensive heart disease, 2.3 from infections and 2 from various respiratory diseases [2,10]. In addition, life expectancy was shortened by more than 10 years among current smokers compared to non-smokers. Adults who quit smoking between ages 25 to 54 gain around 8.5 years of life on average compared with those who continued to smoke [9]. Smoking cessation is also associated with a

considerable and rapid benefit in decreasing the risk of stroke, particularly in light smokers (<20 cigarettes /d) [11]. It is estimated that cigarette smoking costs the US 96 billion in direct medical expense and $97 billion in lost productivity per year [12].

However, there are some side effects of smoking cessation. For example, smokers who attempt to quit, experience nicotine withdrawal symptoms such as headaches, depression and anxiety [13]. In addition, smokers generally gain weight when they quit smoking [14-18]. This is due to the effect of nicotine on the body. Nicotine accelerates the baseline metabolism rate and suppresses appetite. Once it is removed, the baseline metabolism rate decreases and food consumption increases, resulting in excess weight [15]. Using the 2003 - 2012 National Health and Nutrition Examination Survey (NHANES), Veldheer et al. found that the mean 10-year weight gain among continuing smokers was 3.5 kg (7.7 lbs.) versus 8.4 kg (18.5 lbs.) among former smokers after controlling for age, gender, education and race [18]. The potential weight gain associated with smoking can lessen some of the health benefits of quitting.

While most people gain weight after smoking cessation, there is considerable variance in the amount of weight gain. A meta-analysis done by Aubin et al. in 2012 found, from 62 studies, the mean weight gain was 4.67 kg (10.3 lbs) at 12 months after quitting. But around 13% of quitters gained more than 10 kg (22 lbs) [19]. Another meta-analysis done by Tian et al. in 2015 identified 35 cohort studies, including 63,403 quitters and 388,432 smokers. The mean weight gain was 4.10 kg (9 lbs) among quitters, compared with 1.5 kg (3.3 lbs) for continuing smokers [20].

I outline three papers to study the interaction of smoking behaviors and weight. Briefly, the first paper examines the age-specific changes in BMI and BMI distribution among US adults, adjusting for smoking and socioeconomic status (SES). The second study investigates the tradeoff between the health benefits of quitting smoking and the harm due to post-cessation weight gain. Lastly, I explore interventions that could reduce post-cessation weight gain and estimate the potential increase of overall welfare in the population.

Chapter 1: What are the age-specific changes in BMI and BMI distribution among US adults? Does either smoking behaviors or socioeconomic status affect the BMI changes?

While many studies investigated trends in obesity or BMI trajectories, they failed to address the BMI changes across the BMI spectrum or whether a period effect exists across birth cohorts. In the first paper, I examine population representative annual BMI change across two time periods (1997 to 2007 and 2007 to 2017) by age, gender, SES and smoking status using a synthetic cohort approach. I also assess the changes in BMI distribution among age-specific groups. Information on BMI change and BMI distribution change can help us identify age-groups that are more susceptible to excess weight gain.

Chapter 2: Can the harm from post-cessation weight gain completely offset the benefits from smoking cessation?

Using survival analysis, I calculate the tradeoff between smoking cessation and post-cessation weight gain using mean survival time. I estimate the break-even weight gain, i.e. the weight individuals would need to gain to offset the benefits of smoking cessation. A potentially attainable break-even weight gain exists for certain combinations of quit-age and BMI. The break-even

weight gain decreases with quit-age. The break-even weight gain for white males with an initial BMI of 30 is 29.9 kg if they quit at 50 but decreases to 4.2 kg if they quit 80. I identify subgroups with higher initial BMI and higher cigarette consumption as high-risk to receive negative net health benefits from quitting. Compared with smokers who experience no post-cessation weight gain, those who experience an average post-cessation weight gain lose 0.64 life-years saved from quitting. Smoking cessation should be coupled with weight management programs to maximize health gains.

Chapter 3. Can some interventions help smokers quit while reducing post-cessation weight gain? How much health benefits can quitters potentially gain?

In this study, I estimate the health benefits gained from reduction in post-cessation weight gain using an agent-based model. The model simulates three interventions from different categories: pharmacotherapy, physical activity intervention and alternative tobacco product (electronic cigarette). The effectiveness of these interventions is assessed in terms of cumulative survival probability, life-years saved and obesity prevalence at the population level. I first simulate a nationally representative sample of adult smokers in the US via the National Health Interview Survey (NHIS) in 2017. Then interventions affecting both smoking cessation rates and post-cessation weight gain are implemented among the simulated population. Model results show that electronic cigarette is the dominant intervention that increases survival probability and reduces obesity prevalence. If all adult smokers quit smoking and switch to electronic cigarette, they will experience a 1.25 percentage points increase in cumulative survival rate and 3.11 percentage points reduction in obesity prevalence in 20 years.

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Chapter I

Age-Specific Changes in BMI and BMI Distribution Among US Adults Using Cross-Sectional Surveys From 1997 to 2017

1.1 Background

Globally the obesity prevalence is growing at an alarming rate with around 2 billion individuals overweight and one third of them obese [1]. Similar to many countries, the adult obesity prevalence in the US has increased in the past few decades. In 2015, the obesity prevalence was around 40% among US adults, compared with 30% in 1999 [2].

Obesity is a result of excess weight gain from imbalance between energy intake and consumption. It is associated with adverse health conditions such as heart disease, stroke, type2 diabetes and certain types of cancer [3,4]. Studies have shown that obesity affects some groups more than others. For example, obesity is closely associated with age. Adults between the age 40 and 59 tend to have higher obesity prevalence than other age groups [2]. Race is another important factor. Hispanic and non-Hispanic black adults have higher obesity prevalence than non-Hispanic white adults [2, 5]. In addition, obesity prevalence varies by socioeconomic status (SES) and smoking status [6,7]. Individuals with lower income or less education (less than high school) tend to have a higher prevalence of obesity [7]. Smoking initiation is associated with weight loss and smoking cessation with weight gain [8,9].

Some studies have addressed changes in BMI or BMI trajectories over time. For example, Nonnemaker et al. examined seven rounds of the 1997 National Longitudinal Survey of Youth to examine BMI trajectories among youth. They identified the BMI trajectories into four classes based on risk of becoming obese by adulthood and discovered some variables that are potentially associated with class membership such as race, gender and mother's education [10]. Another study using the Quebec Longitudinal Study of Child Development followed 2,210 children for 8 years. Researchers were able to identify three trajectories of BMI. For the group with an increasing average BMI, maternal BMI and maternal smoking during pregnancy are strongly associated with the group [11]. While most studies examined BMI trajectories for children or adolescents [10-14], some also investigated BMI trajectories in adulthood. Peralta et al. assessed association of BMI trajectories from 34 to 54 years with lung function decline. They found that overweight and obese trajectories of BMI decreased lung function [15]. A recent publication by Song et al. examined the influence of genes on BMI trajectory in adulthood. They created a genetic risk score and discovered a nonlinear association between the score and BMI over time [16].

However, most studies focus on trends in obesity prevalence or changes in mean BMI. They do not address BMI changes across the spectrum or whether a period effect exists across birth cohorts [10-12,16]. In addition, previous studies tend to cover a fraction of adulthood instead of examining changes in the BMI distribution over an extended age period [15]. This study contributes to the literature by examining the age- and gender-specific changes in BMI and BMI distribution over adulthood (20-65) and across two time periods (1997- 2007 and 2007-2017). Factors that might affect BMI changes such as SES and smoking behaviors are also assessed. Information on changes

in BMI and BMI distribution can help us identify subgroups that are more susceptible to excess weight gain.

1.2 Methods

I adopted a synthetic cohort approach to examine longitudinal changes in adult BMI and BMI distribution across two time periods: 1997 to 2007, and 2007 to 2017. Synthetic cohort approach constructs a pseudo panel from multiple cross-sectional surveys by matching participants by birth year. It is a popular tool used by econometricians and epidemiologists when there is a lack of panel data [17,18].

Sample

I combined three waves of the National Health Interview Survey (NHIS) to construct the synthetic cohort. NHIS has monitored the health of US population since 1957. It was conducted every year on a nationally representative sample of the population and the data was collected by the US Census Bureau. My sample included survey years 1997, 2007 and 2017 for the synthetic cohort analysis.

The final sample excluded women who were pregnant at the survey, individuals with missing BMI information and individuals under the age of 20. The sample size is 33,645 in 1997, 21,220 in 2007 and 24,981 in 2017, a total of 79,846 observations from three waves of NHIS.

Mean BMI change by age and sex

Mean BMI was determined for each age- and gender-group adjusting for survey design by incorporating survey weights for sample adults. I constructed synthetic cohorts in two time periods to examine longitudinal change of BMI: between 1997 to 2007 (period 1), and between 2007 and 2017 (period 2). Age groups in five-year bands are used to match birth cohorts. For example, individuals who are between 20 and 24 years old in 1997 are matched with individuals between 30 and 34 in 2007. There are a total of 10 cohorts between age 20 and 65 in 1997 who are matched to 10 cohorts between age 30 and 75 in 2007. Similarly, 10 cohorts between age 20 and 65 in 2007 are matched to cohorts between 30 and 75 in 2017. Changes in mean BMI within each period were determined for each synthetic cohort and the standard deviation of the difference between the means was determined by a z-test in STATA.

Age- and gender-specific change in BMI distribution

Another important measure in this study is the age- and gender-specific changes across the BMI distribution. The sample from 2007 was divided into 9 age-groups with 5-year increments by gender and matched with 9 age-groups in 2017 respectively, accounting for the aging effect. For example, men who were 30-34 years old in 2007 are matched with men from 40-44 years old in 2017. Graphs are presented to illustrate the age- and gender-specific longitudinal changes in the BMI distribution over time.

In addition to the BMI distribution, longitudinal changes across the BMI spectrum are also investigated. Variations across the BMI spectrum can be telling of whether the weight gain changes differ based on initial BMI values. For example, individuals with higher BMI entering adulthood

might experience more weight gain in the next 20 years than those with lower BMI. I categorized individuals by gender into 4 age-groups with 10-year increments. These subgroups were further divided into 20 quantiles according to their BMI values. For example, a BMI of 25 in 2007 for 20-29 men was in the 40th quantile. For all these quantiles by age group, I matched them with their corresponding age-groups and quantiles in ten years (1997-2007 vs 2007-2017) to study the changes. If BMI gain cross the spectrum are the same, then we should observe a horizontal line parallel to the x-axis. If we see a line with positive slope, it implies the weight change is higher at the upper end of the BMI spectrum.

Impact of socioeconomic or smoking status on BMI change

Many studies have discovered the weight-related socioeconomic disparities [19-22]. Individuals with high SES are less likely to be obese [20,21]. Obesity prevalence among adults with some college education is around half the prevalence for those who did not finish high school [23]. Many factors contributed to these disparities such as diet, health behaviors and neighborhoods [20,22,24]. For example, teenagers with low SES consumed more fast food than their counterparts from high SES [20,22]. Neighborhoods with few grocery stores create food desserts and limit access to healthy food. Areas where major residents are minority and low-income groups are more likely to have food desserts [24].

These studies suggest the significant impact of SES on obesity prevalence and average BMI in the population, this study examines whether SES affects BMI changes over time as well. SES status is approximated by income where a binary variable is created. Individuals whose household income are above the national median are determined as high SES and the others as low SES.

Median household income is approximated to be $30,000 in 1997 and $50,000 in both 2007 and 2017.

Cigarette smoking also affects body weight. Due to the effect of nicotine, smokers have higher metabolism rates and depressed diets. The former increases energy consumption and the latter reduces food intake, both contributing to lower weight [25-27]. Adult smokers tend to have lower BMIs than never-smokers [28-31]. This study examines whether smoking also affects age- and gender-specific BMI changes by comparing smokers with never-smokers. I first stratified age- and gender-specific groups by smoking behaviors, a categorical variable indicating current smokers or never-smokers, and then determined changes in each age-gender and smoking strata.

The analyses in this study were conducted via STATA v14.0 (StataCorp LP, College Station, TX). Population representative estimates of weight and BMI were calculated via the svy command in STATA that accounts for individual weights and survey design.

1.3 Results

Age- and gender-specific BMI changes

Table 1.1 shows the mean BMI by age and gender for matched synthetic cohorts across two periods: period I from 1997 to 2007 and period II from 2007 to 2017. And Table 1.2 presents the age-specific changes in BMI by gender in period I and period II. Among men and women in both periods, BMI first increases with respect to age, then decreases for the elderly. This non-linear pattern is due to the effect of aging where the elderly experience muscle loss and consequently weight loss [32]. This pattern also implies a non-linear relationship between weight gain and age.

Individuals are expected to experience positive weight gain at the beginning and gradually this weight gain diminishes and becomes weight loss. Controlling for age and gender, mean BMI increases over time. For example, for women age 30-35, their mean BMI grew from 25.31 in 1997 to 27.41 in 2007 and 27.78 in 2017.

Table 1.1 Mean BMI by age and gender for matched synthetic cohorts in across two periods.

Table 1.2 BMI changes by age and gender for matched synthetic cohorts between two periods.

Figure 1.1 shows annualized data for mean BMI change by age over two different periods for men. The x-axis is the baseline age cohort, ranging from 20 to 64 years old. The y-axis is the mean BMI change annually with a 95% confidence interval. The black open squares are for period I (1997-2007) and blue solid squares for period II (2007 – 2017). In general, BMI change is age-dependent with younger men gaining more BMI than the older ones. The changes in BMI do differ across two periods. From 1997 to 2007, men tend to put on more weight over time than their counterparts from 2007 to 2017. For example, for men between 20 and 24 in 1997, they gained an average of 0.26 BMI per year in ten years. But for those between 20 and 24 in 2007, they only gained an average of 0.19 BMI per year in ten years. If we assume an average height of 1.78m, this weight gain is 0.82 kg in period 1 and only 0.6 kg in period 2.

Figure 1.1 Age-specific increase in mean BMI between 1997-2007 and 2007-2017 for men.

The annualized data for mean BMI change by age over two different periods for women can be found in Figure 1.2. Similarly, there is a strong age-dependent BMI change. Younger women

tend to gain more weight and those in period II gains slightly less weight than their counterpart in period I.

Figure 1.2 Age-specific increase in mean BMI between 1997-2007 and 2007-2017 for women.

Age- and sex-specific change in BMI distribution

Figure 1.3 and 1.4 below illustrate the changes in BMI distribution between 2007 and 2017 for 9 synthetic age-cohorts by gender. Solid lines represent BMI distributions in 2007 and dashed ones are BMI distributions in 2017 for the same age cohort. The x-axis is BMI and y-axis the population density. Across all cohorts, the BMI distribution has shifted to the right in ten years and become more right skewed. The shift is the greater for younger men and women. The shifts in BMI distribution for elder men and women are minor. These findings are consistent with previous research where increasing obesity prevalence across age-groups are discovered over time.

Figure 1.3 Change in BMI distribution between 2007 and 2017 by age cohorts for men.

Figure 1.4. Change in BMI distribution between 2007 and 2017 by age cohorts for women.

Figure 1.5 shows the pattern of BMI change across the BMI spectrum. For each sub-graph, the x-axis is the baseline BMI value and the y-axis is the annual change in BMI. The black open circles represent changes in period I and the blue circles represent changes in period II. In all subgraphs, BMI gains do not lie parallel to the x-axis. We observe a growth in BMI when moving to the upper end of the BMI spectrum. These patterns indicate differential weight gains where individuals with higher initial BMI values gain more weight. This pattern holds in both period I and period II. For

example, from 1997 to 2007, 20-29 men in the 10th percentile gained around 0.03 BMI annually while those in the 90th percentile gained 0.015 BMI annually.

Figure 1.5 Change in BMI across the BMI spectrum for men and women over two periods.

Impact of socioeconomic status and smoking behaviors

SES approximated by household income has a significant effect on age-specific changes among men and women. Age-dependent linear regressions for high vs low SES resulted in different slopes for men ($p=0.099$) and women ($p = 0.04$). For both genders, individuals with low SES experience greater variations in BMI gain over time.

Figure 1.6 Age-specific change in BMI for men and women between 2007 and 2017, stratified by household income.

Male smokers tend to experience insignificant weight changes as they age. For example, between the age of 30 and 64, the mean BMI changes are all insignificant. As a result, male smokers have lower BMI than never-smokers with positive weight gains. This pattern is similar for female smokers. They also experience insignificant weight changes between the age of 35 and 64, except for the period of 45 to 49.

Figure 1.7 Age-specific change in BMI for men and women between 2007 and 2017, stratified by smoking behaviors.

1.4 Discussion

This study investigated the age- and gender-specific longitudinal BMI change among US adult smokers from 1997 to 2019 with the help of synthetic cohort analysis. There are a few important findings. First, BMI change is strongly age-dependent. Younger individuals experience greater weight gains. These weight gains gradually diminish over time and after the age of 60 or 70 when individuals start to lose weight as a result of aging and muscle loss. Compared with the period from 1997 to 2007, annual weight gain has stayed pretty similar except for young males, who actually see a slight decrease in weight gain.

In addition to BMI changes over time, I also examined age-specific mean BMI gain across the BMI distribution. There exists differential weight gain across the spectrum with greater gains at the upper end of the BMI spectrum in both periods.

The age-specific mean BMI gain have not increased from period i to period ii. However, mean BMI for each age-cohorts provided in Table 1 suggest otherwise, where there is an increasing BMI for the same age-cohort across time. One plausible explanation is that although BMI changes are relatively constant, initial BMI of individuals entering adulthood have increased. In this case, we would also observe an increasing obesity prevalence across age-cohorts. For example, if males entering adulthood in 1997 with a BMI of 20, they are not going to become obese in the next ten years with an annual increase of 1 BMI per year. However, with the same BMI growth, another man with an initial BMI of 25 would become obese in 5 years.

The relatively constant BMI gain in adulthood across two periods suggests higher weight gain for children or teenagers in the later period. Consistent with this hypothesis, scholars have discovered an increasing rate of childhood obesity in many countries [2,33]. Using the National Health and Nutrition Examination Survey, Hales et al. reported a youth (2-19) obesity prevalence to be 18.5% in 2015, compared with 17.2% in 2013 and a 13.99% in 1999. Childhood or adolescent obesity is also closely connected with adulthood obesity. Obese children and teenagers were five times more likely to become obese in adulthood than those who were not [34]. This finding is crucial in designing public health interventions for obesity. If adult obesity is a result of higher weight when entering adulthood, more efforts should be put to reduce weight gain or preventing obesity for children and adolescents.

The significant impact of SES on weight gains suggest ongoing inequalities where population with lower income suffer from greater weight gains. At the same time, smoking behaviors modify weight gains where smokers experience less weight gain. Smokers tend to concentrate among low SES population. This relationship between BMI and smoking behaviors via SES further complicates the interpretation of population level measures. To some extent, smoking has reduced the weight disparity across low and high SES. If all current smokers quit smoking the next day, we will see an increase in average BMI due to post-cessation weight increase and also a growth of obesity prevalence. The BMI shift will concentrate among minority and lower income groups as well. Consequently, if no weight management program is added to smoking cessation intervention, we might observe an exacerbation of disparities in weight across SES.

There are some limitations to this study. First, smoking behaviors are self-reported data in NHIS where measurement errors might occur. And SES in this study is measured as a binary variable via median household income. Other alternative measures of SES will be included in the future to test the relationship. For example, education attainment and occupations are common measures of SES. Another limitation is the differential mortality rate with respect to BMI values. Mean BMI and BMI changes could be partly due to selection bias where less healthy individuals experience higher mortality rates. For example, extremely low BMI is indicative of severe illness and class III obesity (BMI > 40) is associated with elevated mortality rates. In this study, I assume the trend of differential mortality rates is the same over 20 years. But with the development of technology, heavier and less healthy people are more likely to survive, resulting in increased BMIs across the population.

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Tables and Figures

Table 1.1 Mean BMI by age and gender for matched synthetic cohorts across two periods.

	BMI (kg/m2)											
	1997 NHIS*				2007 NHIS				2017 NHIS			
	Age group	Mean	s.d.	n	Age group	Mean	s.d.	n	Age group	Mean	s.d.	n
Men	20-24	25.4	4.31	1128	20-24	26.22	4.88	816	20-24	-	-	-
	25-29	26.39	4.77	1533	25-29	27.44	5.1	879	25-29	27.54	5.46	955
	30-34	26.5	4.63	1763	30-34	27.99	5.76	946	30-34	28.15	5.57	965
	35-39	27.07	4.67	1797	35-39	28.37	5.26	921	35-39	28.39	5.26	924
	40-44	27.4	4.68	1684	40-44	28.38	5.08	935	40-44	29.27	5.70	841
	45-49	27.42	4.7	1427	45-49	28.58	5.03	991	45-49	29.23	5.47	961
	50-54	27.75	4.64	1175	50-54	28.61	5.55	912	50-54	29.75	5.84	971
	55-59	27.55	4.68	905	55-59	28.40	5.33	789	55-59	28.99	5	1037
	60-64	27.22	4.83	841	60-64	28.34	5.5	717	60-64	29.09	5.88	1109
	65-69	27.13	4.74	778	65-69	27.73	5.69	600	65-69	28.89	6.62	1021
	70-74	-	-	-	70-74	27.53	4.89	438	70-74	28.07	5.25	818
Women	20-24	24.33	5.1	1449	20-24	24.46	4.85	941	20-24	-	-	-
	25-29	24.79	5.77	1838	25-29	25.81	6.25	986	25-29	27.17	6.39	982
	30-34	25.31	6.03	2055	30-34	27.41	7.35	1024	30-34	27.78	7.08	1071
	35-39	25.56	5.76	2226	35-39	27.39	7.56	1091	35-39	28.36	7.03	1046
	40-44	26.24	5.97	1932	40-44	27.64	7.12	1075	40-44	29.04	7.18	934
	45-49	26.53	5.79	1692	45-49	27.08	5.95	1122	45-49	28.44	6.43	982
	50-54	27.11	5.92	1363	50-54	28.02	7.27	1068	50-54	28.48	6.38	1155
	55-59	27.1	5.65	1106	55-59	28.26	6.47	890	55-59	28.88	7.69	1177
	60-64	26.89	5.57	968	60-64	28.11	6.48	808	60-64	28.64	7.81	1224
	65-69	26.4	5.61	1092	65-69	27.93	6.61	680	65-69	28.49	7.74	1216
	70-74	-	-	-	70-74	27.38	6.47	546	70-74	28.02	7.16	994

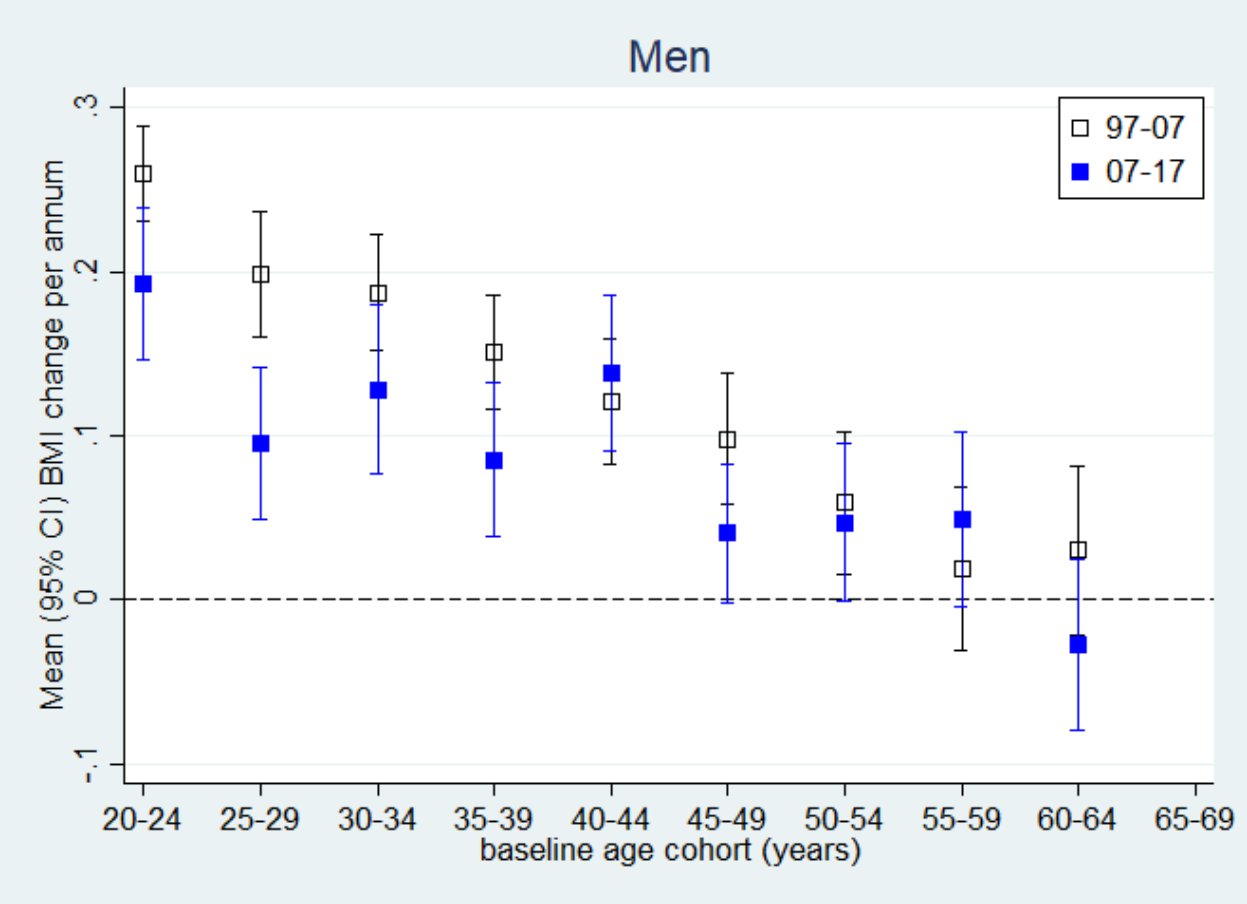
*Note. * NHIS: National Health Interview Survey*

Table 1.2 BMI changes by age and gender for matched synthetic cohorts between two periods.

	BMI (kg/m^2)					
	Period I			Period II		
	Age group	Mean	s.d.	Age group	Mean	s.d.
Men	20-24	2.6	0.15	20-24	1.93	0.24
	25-29	1.98	0.19	25-29	0.95	0.24
	30-34	1.87	0.18	30-34	1.28	0.26
	35-39	1.51	0.18	35-39	0.85	0.24
	40-44	1.21	0.19	40-44	1.38	0.24
	45-49	0.98	0.21	45-49	0.41	0.21
	50-54	0.59	0.22	50-54	0.47	0.25
	55-59	0.18	0.25	55-59	0.49	0.27
	60-64	0.3	0.26	60-64	-0.27	0.27
Women	20-24	3.08	0.26	20-24	3.32	0.28
	25-29	2.6	0.26	25-29	2.55	0.3
	30-34	2.33	0.22	30-34	1.63	0.34
	35-39	1.51	0.25	35-39	1.05	0.32
	40-44	1.78	0.26	40-44	0.84	0.3
	45-49	1.73	0.27	45-49	1.81	0.3
	50-54	1	0.28	50-54	0.63	0.33
	55-59	0.83	0.3	55-59	0.22	0.32
	60-64	0.49	0.33	60-64	-0.09	0.33

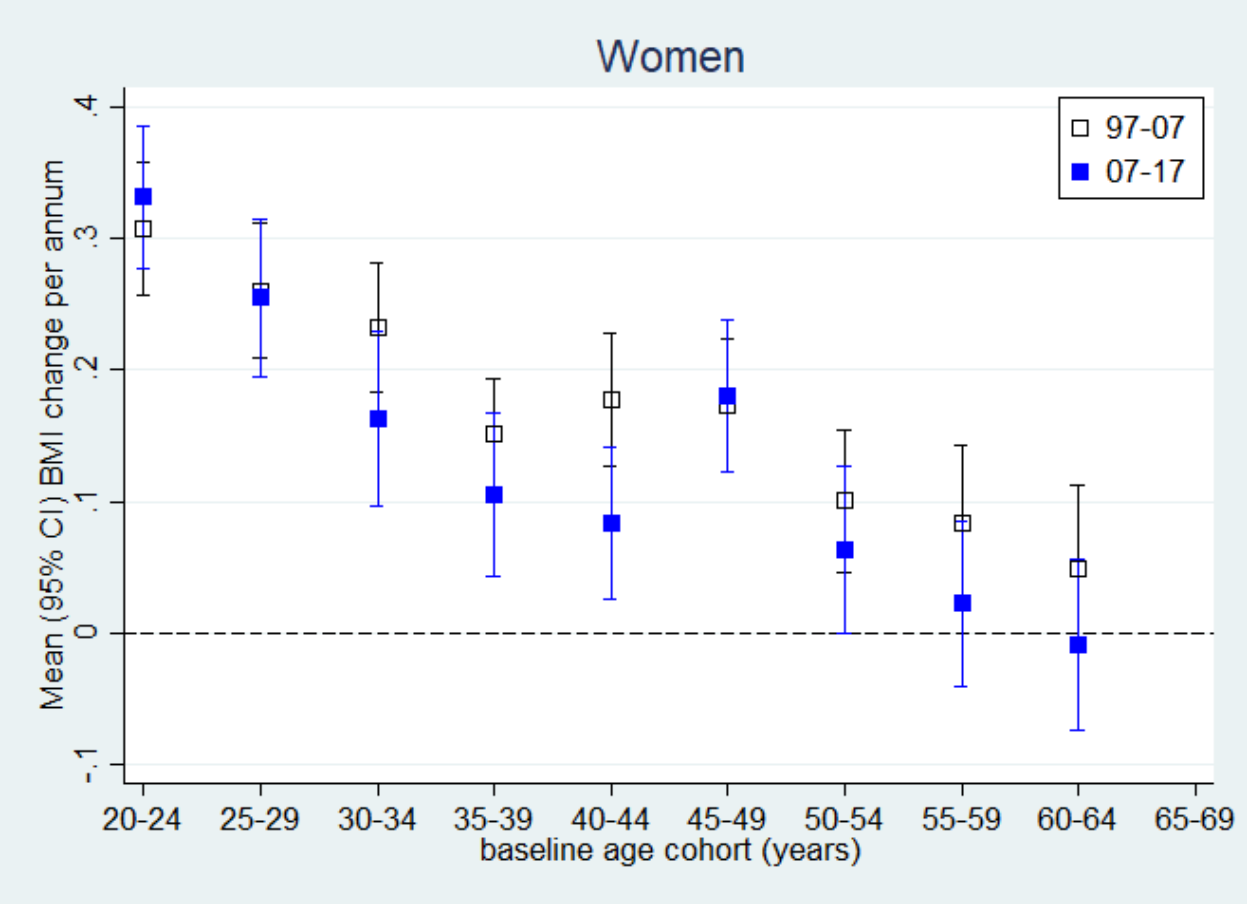
Note. Age group listed are the ages in baseline year. Period I: 1997 to 2007. Period II: 2007 to 2017.

Figure 1.1 Age-specific increase in mean BMI between 1997-2007 and 2007-2017 for men.



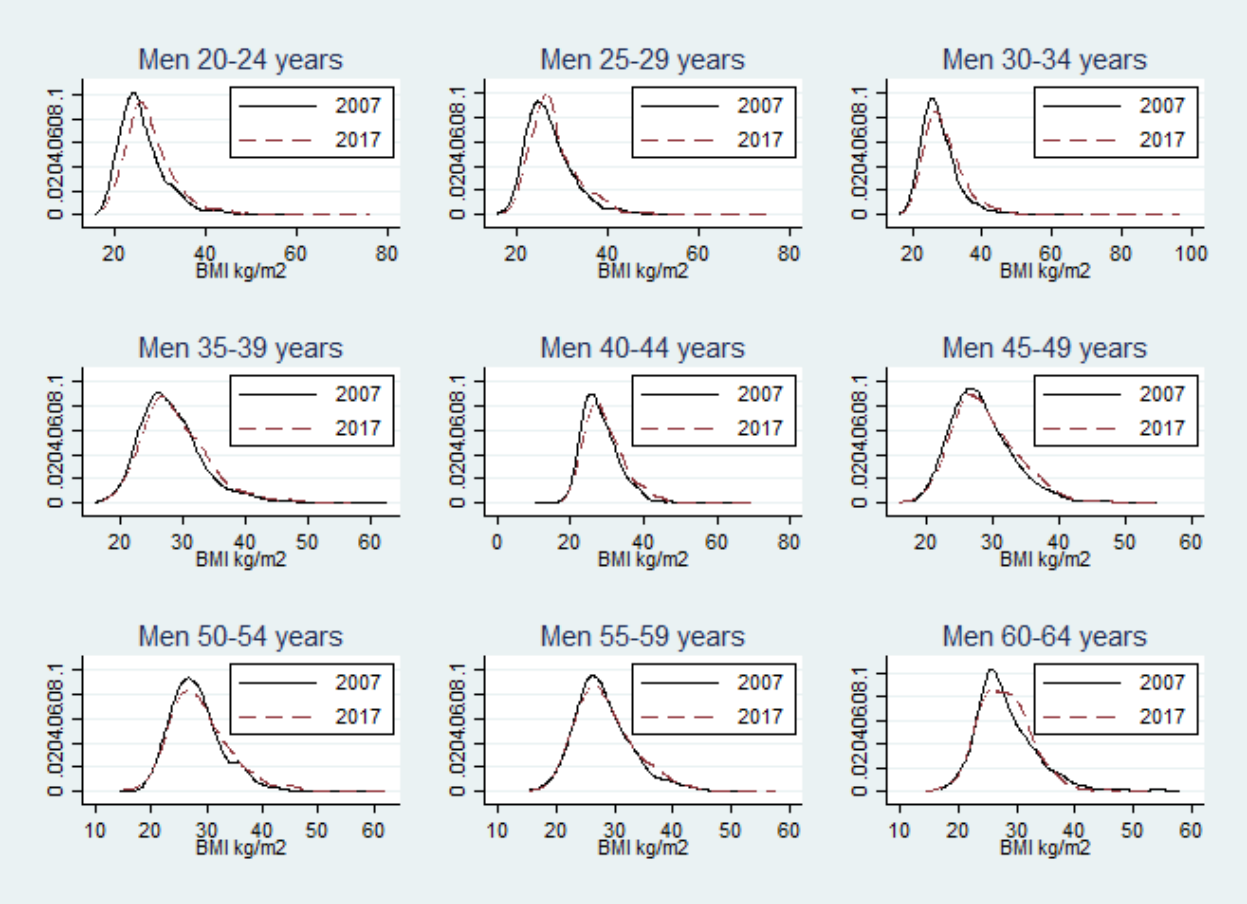
Note. Black open squares: mean BMI change in period I (1997-2007). Blue filled squares: mean BMI change in period II (2007-2017).

Figure 1.2 Age-specific increase in mean BMI between 1997-2007 and 2007-2017 for women.



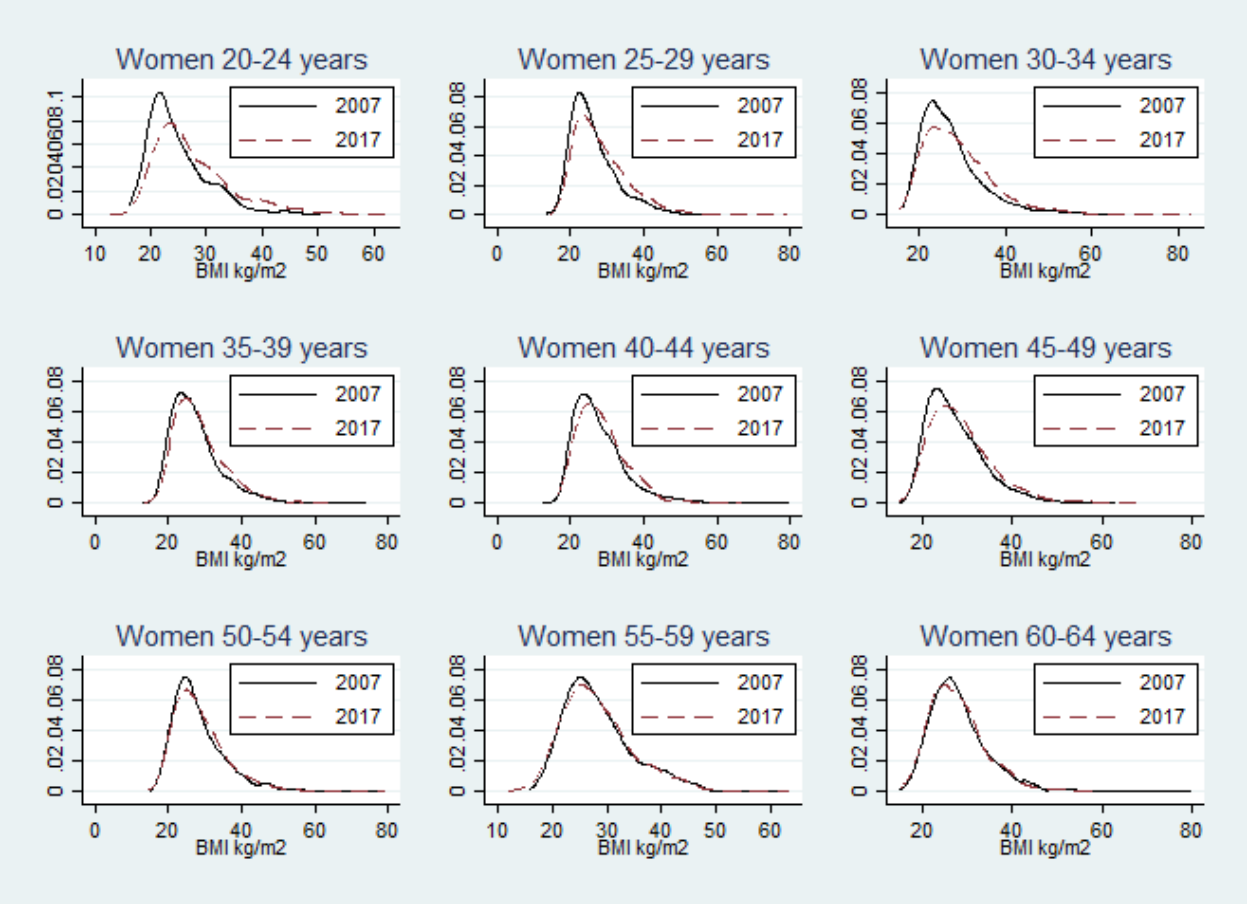
Note. Black open squares: mean BMI change in period I (1997-2007). Blue filled squares: mean BMI change in period II (2007-2017).

Figure 1.3 Change in BMI distribution between 2007 and 2017 by age cohorts for men.



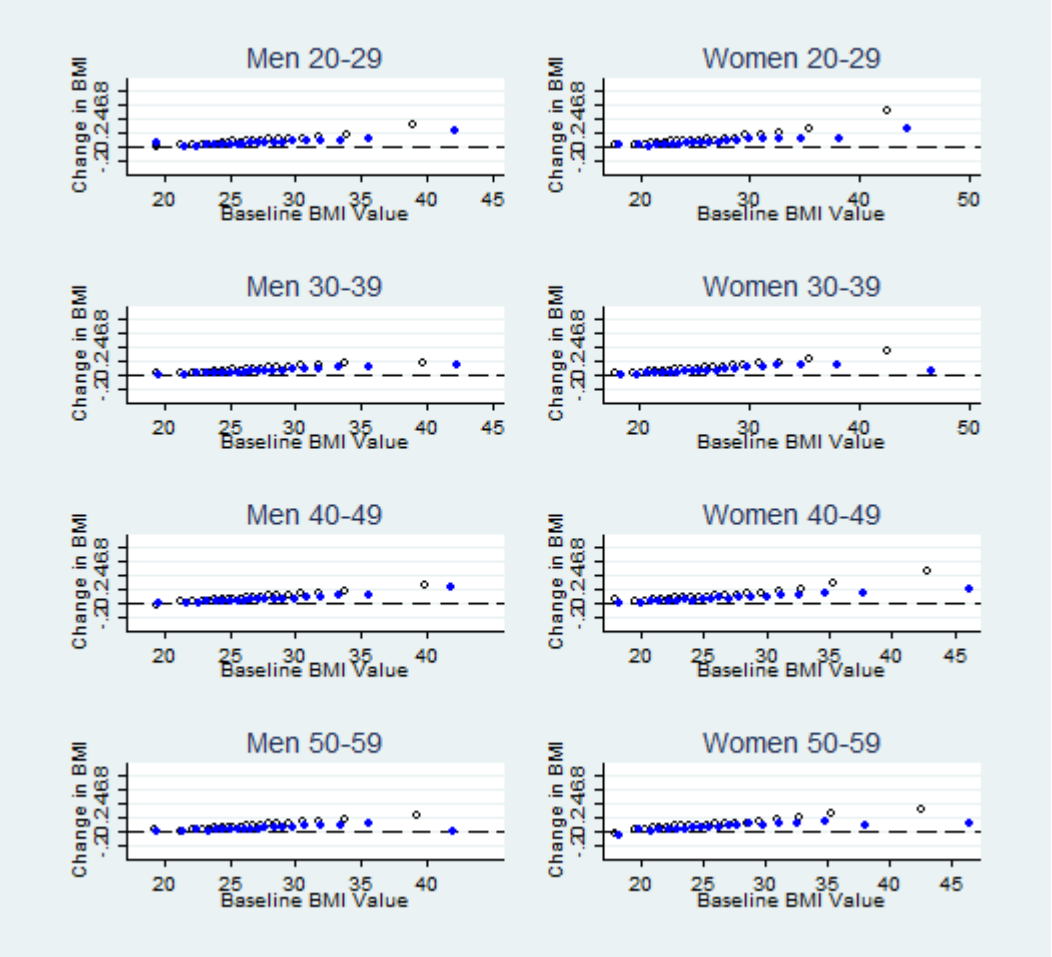
Note. Solid curve: BMI distribution in 2007. Dashed curve: BMI distribution in 2017.

Figure 1.4 Change in BMI distribution between 2007 and 2017 by age cohorts for women.



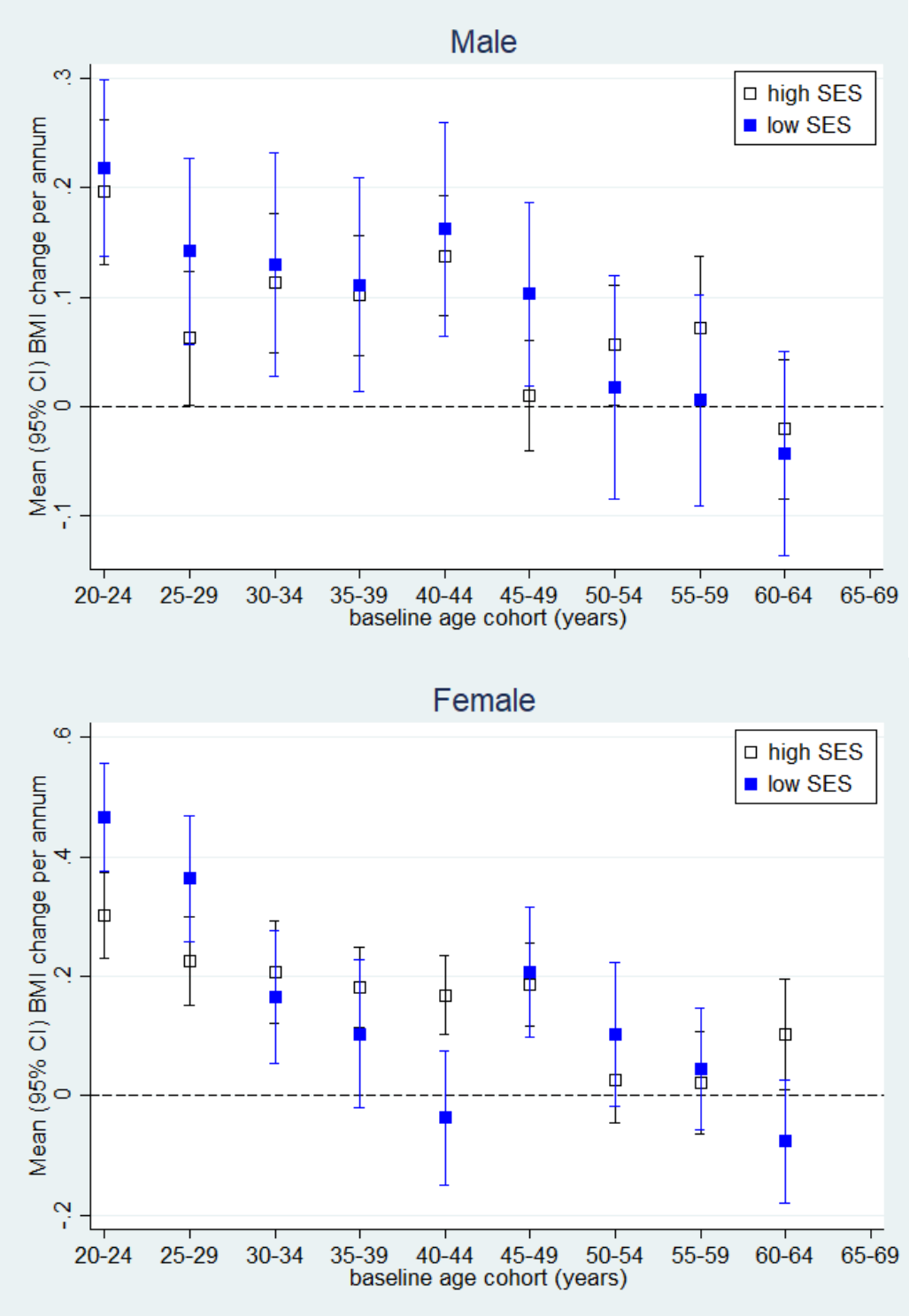
Note. Solid curve: BMI distribution in 2007. Dashed curve: BMI distribution in 2017.

Figure 1.5 Change in BMI across the BMI spectrum for men and women over two periods.



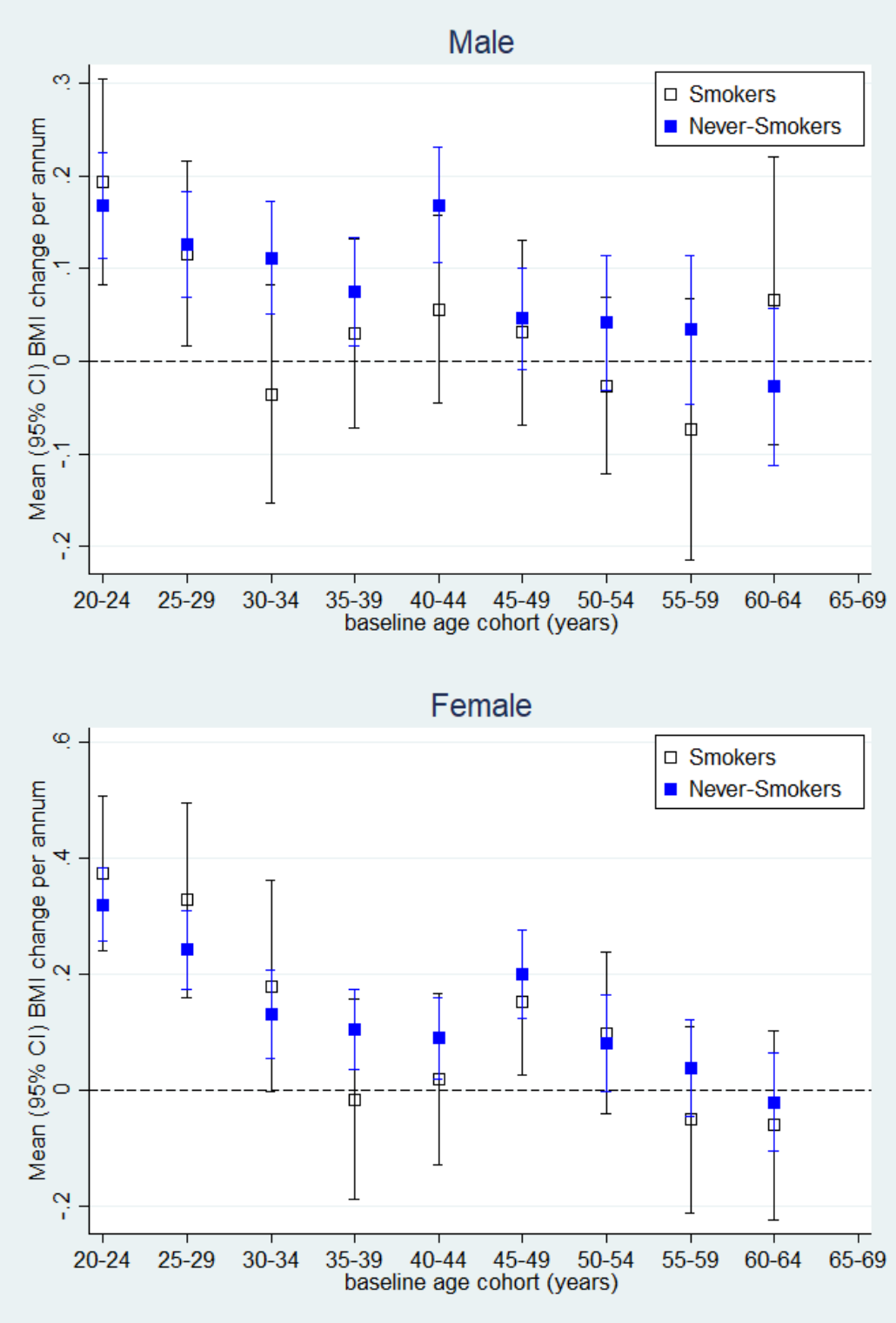
Note. Black open dots: change in BMI in period I (1997-2007). Blue filled dots: change in BMI in period II (2007-2017).

Figure 1.6 Age-specific change in body mass index for men and women between 2007 and 2017, stratified by household income.



Note. Blue filled squares: low SES with less than median household income; black open squares: high SES with greater than median household income.

Figure 1.7 Age-specific change in body mass index for men and women between 2007 and 2017, stratified by smoking behaviors.



Note. Blue filled squares: current smokers; black open squares: never-smokers.

Chapter II

Comparing Benefits of Quitting vs Harm Due to Post-Cessation Weight Gain: Evaluating Potential Break-Even Scenarios

2.1 Background

Smoking cessation has well-documented health benefits. Studies have found that the all-cause mortality rate is about three times among smokers compared to those who have never smoked [1]. Despite the health benefits associated with smoking cessation, there is an unintended consequence of quitting: post-cessation weight gain. This weight gain can lessen some of the health benefits of quitting. For example, post-cessation weight increase contributes to an increased risk of type 2 diabetes in the short run, hypertension, and a reduced improvement of lung function [2,3]. Furthermore, post-cessation weight gain serves as a major deterrent for smokers to quit, especially female smokers who are concerned about their body image [4-6]. In a national random-digit dialing survey of women smokers, more than 60% described themselves as somewhat concerned about post-cessation weight gain. The authors concluded that weight-concerned women will be unlikely to seek smoking cessation treatment due to self-image issues [4]. Similarly, in a study done in the Czech Republic, around 34% of all patients in a smoking cessation treatment were concerned about weight (19.4% for men and 49.7% for women). These weight concerns were associated with delay in quitting [6].

The physiological mechanism that smoking affects body weight is through nicotine. Nicotine is known to increase metabolism and decrease body weight. Body weight is determined by the difference between calorie intake from food and daily energy expenditure, which can be further divided into subcategories of basic metabolism rate, physical activity and thermic effects of food [7,8]. Nicotine reduces body weight by increasing basic metabolism rate as well as suppressing appetite that results in less food intake [9,10]. Once smokers stop smoking, their metabolism rates slow down and their appetites increase, leading to weight gain.

While most people gain weight after smoking cessation, there is considerable variance in the amount of weight gain. A meta-analysis done by Aubin et al. in 2012 found, from 62 studies, the mean weight gain was 4.67 kg (10.3 lbs) at 12 months after quitting. But around 13% of quitters gained more than 10 kg (22 lbs) [11]. Another meta-analysis done by Tian et al. in 2015 identified 35 cohort studies, including 63,403 quitters and 388,432 smokers. The mean weight gain was 4.10 kg (9 lbs) among quitters, compared with 1.5 kg (3.3 lbs) for continuing smokers [12].

However, most of these studies reported in the systematic reviews only followed individuals up to 12 months after cessation. Only a few studies reported weight gain over the long term in biochemically confirmed abstainers. The Lung Health Study found quitters gained 8.2 kg (18.1 lbs) over 5 years, versus the 1.6 kg (3.5 lbs) gained by smokers [13]. Lycett et al. followed a total of 1686 participants for 8 years in 19 general practices in Oxfordshire, UK. Abstainers gained 8.79 kg (19.4 lbs) where smokers gained 2.24 kg (4.9 lbs) on average at the end of 8 years [14]. Since majority of the literature measured only weight changes in smokers vs quitters as point estimates, it remains unclear whether this weight gain is permanent or diminishes over time. One study that

investigated weight trajectory is from Williamson et al. using the First National Health and Nutrition Examination Survey (NHANES I, 1971 to 1975). They found that smokers gain on average, 4.5 kg (9.9 lbs), within 6-12 months after quitting. And their weight returned to the same weight-age trajectory as that observed in never smokers in around 10 years [15]. However, a recent paper using the Framingham Heart Study showed that post-cessation weight gain is still detectable after 20 years of quitting [16].

Researchers have discovered that the benefits of smoking cessation also differ depending on quit-age, aka at what age smokers quit smoking [1,17]. For example, adults who quit smoking between 25 and 34 gain around 10 years of life expectancy while those who quit between 45 and 54 only gain 6 years on average [1]. Based on a study of women in the UK, those who quit before age 40 years avoided more than 90% of the excess mortality caused by continuing smoking [17].

Despite the health gains from smoking cessation, post-cessation weight gain can attenuate the health benefits of quitting. Since the benefits of smoking cessation decrease with age-at-quit (quit-age), it is conceivable that the benefits obtained by quitting at a later age could be completely offset by the harm associated with the weight gain. In this study, I estimate the break-even weight gain, i.e. the minimum weight individuals could gain to completely offset the benefits of smoking cessation, adjusting for age, gender, race and initial BMI. I also look for crucial factors that affect the break-even weight gain, such as quit-age, gender and initial BMI. High risk groups, those who are likely to put on post-cessation weight that is greater than break-even weight gain, are identified. The findings show the perils of addressing these two major public health problems independently

and could be used to parameterize simulation models aimed to investigate the impact of combined policies to address smoking and obesity simultaneously.

This paper is the first study to define and examine the notion of break-even weight gain. It contributes to the ongoing debate on how weight gain after smoking cessation attenuates the health benefits of quitting. It also fills the gap in the literature by directly comparing the benefits of quitting with the harm due to post-cessation weight increase, adjusting for demographic characteristics. It is crucial to study break-even weight gain for smokers to ensure that they receive positive net health benefits.

2.2 Methods

This study is comprised of two major components: 1. Estimating all-cause mortality as a function of BMI and smoking status; 2. Estimating baseline lifetime weight trajectories. First, I calculated the relative risk (RR) for all combinations of BMI and smoking status adjusting for demographic variables. Then I estimated the all-cause mortality rate for normal-weight never-smokers controlling for age, gender and race. Lastly, the baseline lifetime weight trajectory is calculated for current, former and never smokers adjusting for age, gender, race, previous-year BMI, and socioeconomic status (SES). I implemented two forms of post-cessation weight gain for quitters: permanent weight-gain (an instantaneous permanent weight gain following smoking cessation) and diminishing weight-gain (an initial weight gain following smoking cessation which declines over time). Combining these components with discounted life-years saved due to smoking cessation from the survival curve, I computed the break-even weight gain for smokers given gender, race, quit-age, SES and initial BMI.

Relative risk of BMI and smoking status

An article published in the Lancet by the Global BMI Mortality Collaboration assessed the impact of BMI on all-cause mortality using 239 prospective studies in four continents. Using a total of 10,625,411 participants in Asia, Australia and New Zealand, Europe, and North America, the authors concluded that all-cause mortality was minimal at BMI from 22.5 to 25, and mortality increased approximately log-linearly with BMI after 25 [18]. Using their hazard ratio (HR) estimates for various BMI levels controlling for gender, I calculated the impact of post-cessation weight gain for quitters. For BMI over 25, the mortality rate increases log-linearly, adjusting for gender [18]. The functions calculating RR of BMI by gender is listed below:

$$RR_{BMI,male} = \begin{cases} 1.51 & BMI \in (0,18.5) \\ 1.09 & BMI \in [18.5\ ,20) \\ 1.01 & BMI \in [20\ ,22.5) \\ 1 & BMI \in [22.5\ ,25) \\ 1 + \dfrac{(BMI - 25) * 8.6}{100} & BMI \in [25\ ,\infty) \end{cases}$$

$$RR_{BMI,female} = \begin{cases} 1.51 & BMI \in (0,18.5) \\ 1.09 & BMI \in [18.5\ ,20) \\ 1.01 & BMI \in [20\ ,22.5) \\ 1 & BMI \in [22.5\ ,25) \\ 1 + \dfrac{(BMI - 25) * 5.6}{100} & BMI \in [25\ ,\infty) \end{cases}$$

To calculate the RR of mortality adjusting for smoking behaviors, I employed the all-cause mortality RR calculated in Mendez and Warner's work [19]. They analyzed the Cancer Prevention Study II (CPS II) data to derive the RR for men and women current and former smokers as a function of age and, for former smokers, years quit via logistic regressions. Their study showed that the benefits of smoking cessation vary by age and years quit. The later one quits smoking, the less benefits he/she would recover. The equation they used is shown below where i stands for gender (male; female) and j for smoking status (never smoker; current smoker; former smoker).

$$log(RR_{smoking}) = \alpha_{i,j} + \beta_{i,j} \times age + \gamma_{i,j} \times yearquit$$

All-cause mortality rate

Another important piece is the all-cause mortality rate for the base group. Given the all-cause mortality RR of smoking and BMI, I need to calculate the baseline mortality risk to recover mortality rates for all groups. In Mendez and Warner's work, the baseline risk used is the mortality rate for never-smokers [19]. For obesity related RR, the HR is 1 for normal weight individuals with a BMI between 18.5 and 25. Based on these references, the control group in this study are the normal-weight never-smokers, adjusting for age, gender and race.

From a recent publication of the National Vital Statistics Reports, I obtained annual mortality rates in the US by race and gender in 2016 [20]. I then combined the mortality rates with a group prevalence calculated from the National Health Interview Survey (NHIS) in 2016, adjusting for smoking and obesity status. I divided the NHIS sample into 5-year age groups from 40 to 85 and assumed the population prevalence remain the same in these five years. Table 2.1 presents the group prevalence adjusted for smoking and weight statuses by gender, race and age. Individuals are also categorized into one of the nine smoking- and obesity-status groups by gender. There are three groups based on smoking status: 1. Current smoker; 2. Former smoker; 3. Never smoker. And three groups based on obesity status: 1. Overweight (BMI > 25); 2. Normal weight (18.5<BMI< 25); 3. Underweight (BMI< 18.5). Together with the RR of smoking and BMI, I estimated the annual mortality rates in 2016 for all individuals over 40 years old, by one-year increment and adjusted for gender and race [18, 21]. A more detailed description of RRs implemented in the model can be found in Table 2.2. The calculations are conducted using the following formula for each age group:

$$MR_n = \sum_{j=1}^{j=3} \sum_{i=1}^{i=3} RR_{i,j,n} \times W_{i,j,n} \times b_n$$

Here MR is the population level mortality rate in 2016 and n stands for the race- and gender-group. There are four race- and gender-groups: white male; white female, black male and black female. i is smoking status and j is obesity status. There are three possible status for smoking and obesity, respectively. $RR_{i,j}$ is the relative risk of mortality for a race- and gender-group with smoking status i and obesity status j. These relative risks are derived from previous literature. Then I have $W_{i,j,n}$ to be the group specific weight in the population for individuals with smoking status i and obesity status j. Finally, b_n stands for the base group mortality rate for a specific race and gender-group, which is the value I want to calculate.

Table 2.1. Group Prevalence adjusted for smoking and weight statuses by gender, race and age.
Table 2.2. Relative risk (RR) adjusted for smoking and weight statuses by gender.

Table 2.3 below illustrates the all-cause mortality rate estimated for all four race and gender-groups from age 40 till 100, where I assumed the maximum life-span is 100 years. I counted the impact of smoking and obesity on all-cause mortality as additive per the results from Mehta and Preston's work [22].

Table 2.3. Age-specific annual mortality rates for normal-weight never-smokers by gender and race.

Lifetime weight trajectory

The lifetime weight trajectories for current and never smokers are calculated, adjusting for age, gender, race, BMI from previous survey, and SES. I used 12 waves of the Health and Retirement

Study (HRS) from 1992 to 2014. HRS is a longitudinal panel study that surveys a representative sample of the US population every two years. It is conducted by the Survey Research Center at the University of Michigan and sponsored by the National Institute on Aging. At the baseline in 1992, a total of 12,652 individuals from 7,702 households participated. My sample includes only this initial HRS cohort who joined the study in 1992. A few exclusion criteria are applied to obtain my final sample. The flowchart below in Figure 2.1 illustrates the process. I tracked all twelve waves of data (1992 till 2014) to investigate how BMI changes with respect to smoking behavior and demographic variables over time.

Figure 2.1 Flowchart for selecting eligible participants in the final sample from the Health and Retirement Study.

My final sample includes 9,606 unique individuals and 69,745 person-year observations. At baseline, the average age is 55.22 years, 43.04% of participants are male, 80.23% of them are white and the average BMI is 26.99. The obesity rate is 22.93%, 81% are married and around 70% have degrees of high school or above. See Table 2.4 for more details. This sample is referred to as Model I. I also included two other samples with alternative exclusion criteria for robustness check. Compared with exclusion criteria in Model I, Model II kept observations with inconsistent year-quit information while Model III kept observations who passed away within five years of the survey or experienced rapid weight changes.

To estimate the lifetime weight trajectory of individuals adjusting for smoking behaviors and demographic information, I ran multiple linear regressions to predict the percentage change of BMI between any two consecutive surveys adjusting for individual level clustering. I used STATA

14.0 (StataCorp LP, College Station, TX) to conduct this analysis. My dependent variable is the percentage change of BMI between any two consecutive surveys and my independent variables include age, gender, race, SES, and BMI from previous survey. The regression was conducted separately for current and never smokers. Here current smokers are defined as those who report to have ever smoked cigarettes and are currently consuming cigarettes. Never-smokers are those who report to have never smoked cigarettes and are not currently smoking. Regression results for Model I, Model II and Model III can be found in Table 2.5.

Table 2.4 Baseline characteristics of the sample population, Health and Retirement Study 1992.

Table 2.5 Regression results for change in BMI (%) under Model I, Model II and Model III.

Weight-gain mechanism

I implemented two forms of post-cessation weight gain for quitters: permanent weight-gain vs diminishing weight-gain. While many studies have found a permanent weight gain of quitters compared to smokers over an extended period [13,14], a few also found that quitters initially gain weight, but over time their weight trajectory approaches the same age-weight trajectory of never-smokers [8,15]. Permanent weight-gain is an instantaneous permanent weight gain following smoking cessation compared to the age-weight trajectory of continuing smokers with the same initial BMI. The weight difference will remain the same over time. Diminishing weight-gain involves an initial weight gain following smoking cessation and the weight trajectory approaches those of never-smokers in ten years.

Figure 2.2 provides an example to show how life-time BMI trajectories change with respect to one-time smoking behavior change. For a 50-year-old white male with a BMI of 25, he could be

either a smoker or a never-smoker. If he was a smoker, he could either quit and experience post-cessation weight increase at age 51, or he could continue smoking. If he continues smoking, then his BMI is going to follow the blue curve for current smoker. If he quits smoking, he either experiences a permanent post-cessation weight gain (grey curve) or a diminishing weight increase (yellow curve). He could also be a never-smoker with a life-time BMI trajectory shown by the orange curve. The BMI trajectory for former smoker with diminishing weight-gain gradually approaches the trajectory for never smoker. The two curves overlap after age 60 (yellow and orange).

Figure 2.2 Simulated life-time BMI trajectory for a white male with an initial BMI of 25 at age 50, assuming different smoking behaviors.

Break-even weight gain

Employing RRs for smoking and BMI, all-cause mortality for non-obese never-smokers, and the survival curve until age 100, I calculated the break-even weight gain controlling for age, gender and initial BMI. Here initial BMI is the BMI of the quitter at the time when he/she quit smoking. The survival analysis is implemented using Python.

I derived the break-even weight gain adjusting for age and gender with the help of the survival curve. By going over a possible range of weight gains, I looked for the break-even weight gain that completely offsets the discounted benefits gained from smoking cessation examining the discounted total life-years saved till age 100. Discounting rate is set at 3% in the main results and sensitivity analyses are conducted for discounting at1% and 7% [23]. I programmed in Python to conduct this analysis.

Figure 2.3 below is an example of how survival analysis is carried out to calculate the break-even weight gain. Each data point represents the cumulative survival probability for the individual at a certain age. Current smoker (blue) in Figure 3 continues smoking from age 50 till 100. Former smoker I (orange) gave up smoking at age 50 and gained no post-cessation weight while former smoker II (grey) quit smoking also at age 50 but gained a significant amount of weight. I compared whether the area under the survival curve for former smoker is greater than the area under the curve for current smoker, aka a comparison of mean survival time. Since the survival curve for current smoker (blue) lies below the curve for former smoker I (orange), smoking cessation in this case increases mean survival time. However, the survival curve for current smoker (blue) lies above the curve for former smoker II (grey), implying smoking cessation in this context decreases the survival time. As a result, the break-even weight gain lies somewhere between the post-cessation weight gain for former smoker I and former smoker II.

Figure 2.3 Survival curves for current smoker vs former smoker I vs former smoker II from age 50 to 100.

2.3 Results

The average break-even weight gain for individuals with high SES under permanent weight-gain are shown in Figure 2.4. Since there is not much variation in break-even weight gain across initial BMI, Figure 4 presents the average break-even weight gain of initial BMI from 25 to 40. A more detailed description of break-even weight gain by initial BMI can be found in Appendix 2.A. At quit-age of 50, the break-even weight gain for white males is around 9.7 units of BMI, 11.6 units for white females, 9.9 units for black males and 11.4 units for black females. If they quit at 80, the break-even weight gain decreases to 1.41 units, 1.57 units, 1.42 units and 1.59 units, respectively. On average, females have higher break-even weight gain in units of BMI than males at the same

quit-age. Break-even weight gain decreases significantly by quit-age but does not vary much by race.

Figure 2.4 Average break-even weight gain (BMI) by gender, race and quit-age under permanent weight gain.

Figure 2.5 displays the break-even weight gain for all race and gender-groups under diminishing weight-gain at quit-age 80 with varying initial BMI. The break-even weight gain for smokers who quit before age 80 is more than 50 lbs on average; I do not consider that a 50 lbs post-cessation weight gain is highly like and thus omit it in the exhibits. I find that for all race and gender-groups, as initial BMI increases, break-even weight gain decreases. Break-even weight gain decreases from 30.5 lbs at 25 BMI to 12.3 lbs at 40 BMI for white males of average height, 27.6 to 12.1 lbs for white females, 28.8 to 9.4 lbs for black males and 28.4 to 9.5 lbs for black females. I used 1.76 m (5 feet 8) as the male average height and 1.63 m (5 feet 3) as the female average height. Initial BMI is negatively correlated with break-even weight gain for all gender and race-groups controlling for quit-age. Consistent with findings under permanent weight gain, the later one quits smoking, the lower the break-even weight gain is.

Figure 2.5 Break-even weight gain (lbs) at quit-age 80 by gender, race and initial BMI under diminishing weight-gain.

Figure 2.6A shows the total life-years saved for white males with an initial BMI of 25 while Figure 2.6B presents the one for white males with an initial BMI of 40 across various quit-ages. In Figure 2.6A, a white male quits at 50 with no post-cessation weight gain acquires around 4.3 life-years and only 3.6 life-years if he experiences an average post-cessation weight gain of 10.3 lbs (a 16.28% reduction). At quit-age 80, this white male only gains 0.4 life-years with no post-cessation weight

gain and 0.2 life-years with average post-cessation weight gain (a 50% reduction). Post-cessation weight gain reduces the life-years saved from smoking cessation. Similar patterns hold with an initial BMI of 40 in Figure 2.6B.

Figure 2.6 Total life-years saved for white males with no post-cessation weight gain vs average post-cessation weight gain with known initial BMI at age 50.

In addition, I derived life-years saved at the population level if smokers gain zero weight post-cessation versus gaining an average post-cessation weight of 10.3 lbs. Based on the nationally representative smoker sample in NHIS 2017, I estimated 29,186,912 smokers with survey weighting. I excluded observations with missing data and who were pregnant at the time of the survey. I then modified the original Python program to incorporate individual level weight gains to calculate life-years saved for smokers. If all of these smokers quit the next year and experience zero post-cessation weight gain, total life-years saved is estimated to be 150,051,770, that is around 5.14 life-years per smoker. If they experience an average post-cessation weight gain, then total life-years saved will drop by 18,661,067 in total, aka a 0.64 life-year difference per capita.

The results above are for individuals with high SES. I also calculated the break-even weight gain for smokers with low SES under permanent and diminishing weight gain (See Appendix 2.B). In general, the break-even weight gain for individuals with low SES is similar to the ones with high SES. Break-even weight gain calculated using alternative forms of lifetime weight trajectory (Model II and Model III) can also be found in Appendix 2.C. The results are similar to the ones calculated via Model I.

2.4 Sensitivity Analysis

For sensitivity analysis, I first checked the results using discounting rates of 1% and 7%, respectively [23]. Then I reexamined the model by replacing the 2016 mortality rates for normal-weight never-smokers with mortality rates from other years. Sensitivity analysis yielded similar results as main findings, indicating that the results are robust. More details can be found in Appendix 2.D.

2.5 Discussion

This study offers important findings. First, a potentially attainable break-even weight gain does exist for certain combinations of quit-age and BMI. Smoking cessation is beneficial at any age, but post-cessation weight gain could significantly reduce these health benefits.

Second, the break-even weight gain decreases with quit-age. There exists a cut-off age where smoking cessation might lead to more harm than good to smokers. Quit-age is the most important factor affecting the tradeoff between cessation benefits and harm from post-cessation weight gain. Given that quitters gain an average of 10.3 lbs and more than 10% of them gain over 22 lbs [11], a smoker who quits at 80 will have a higher chance to receive negative health benefits from quitting. Currently, the health message provided to smokers is that it is never too late to quit, without addressing the weight concern that many have. The findings show the perils of addressing obesity and smoking independently. Smokers should couple smoking cessation with weight management programs, especially those who might experience a substantial weight gain post-cessation compare to the break-even weight gain.

Results in this study agree with previous findings in general. For most smokers, quitting brings positive net health benefits despite post-cessation weight gain. However, the low break-even weight gain for some subgroups suggests a possibility of negative net health benefits. It is crucial to distinguish smokers who might gain a substantial amount of weight from quitting. A few high-risk groups can be identified based on our study and previous research. The first high-risk group consists of heavy smokers. Studies have shown a positive relationship between post-cessation weight gain and smoking intensity [24]. Heavy smokers (>24 cigarettes daily) tend to put on significantly more weight than either continuing smokers or former light smokers (<15 cigarettes daily) [24]. Another high-risk group are obese smokers. Since higher initial BMI is associated with lower break-even weight gain, obese smokers are likely to gain weight that is above the threshold. Individuals with low SES are more likely to be heavy smokers and suffer from obesity [25,26]. They tend to smoke cigarettes more and for a longer duration [25]. For example, individuals who enroll in Medicaid are about twice as likely as the general population to smoke [27]. Smokers with low SES also have a lower cessation rate on average [28]. Smoking cessation with no weight management for groups with low SES is likely to exacerbate existing health disparities.

Another take-away is the importance to study long-term post-cessation weight gain. The two weight gain mechanisms implemented lead to different values of break-even weight gain. If post-cessation weight gain remains permanent, the break-even weight gain will be lower across all gender and race-groups. We need more research to determine whether post-cessation weight gain diminishes in the long term or not.

One key policy implication is to search for effective weight management programs to help smokers deal with post-cessation weight gain. Existing approaches, such as antismoking medications or weight-related behavioral interventions, have not shown much success, especially in the long run [29-31]. One alternative, electronic cigarette has revealed some promising results. Post-cessation weight gain is significantly decreased when smokers quit or reduced substantially their cigarette consumption by switching to electronic cigarettes [32]. More research is needed to provide further evidence.

There are some limitations of this study. First, the impact of SES on break-even weight gain is underestimated since it is only included in the calculations for lifetime weight trajectory, but not for mortality rates or RRs. Findings in this study show that break-even weight gain does not vary much across SES, but more research is needed to explore this topic. Another limitation is the lack of information on age-specific post-cessation weight gain. Smokers usually quit in their late 40s or early 50s [33]. Future research is needed to investigate the age-specific post-cessation weight gain to derive more accurate results. For the population level estimation of life-years saved, I excluded some observations with missing data. I do not detect any bias in the missing data.

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Tables and Figures

Table 2.1. Group Prevalence adjusted for smoking and weight statuses by gender, race and age.

Age	Overweight Smoker	Under-weight Smoker	Normal-weight Smoker	Overweight Never-Smoker	Underweight Never-Smoker	Normal-weight Never-Smoker	Overweight Former Smoker	Underweight Former Smoker	Normal-weight Former Smoker
White Male									
40-44	0.17	0	0.04	0.4	0.001	0.14	0.21	0	0.04
45-49	0.12	0.001	0.04	0.48	0.008	0.1	0.2	0	0.04
50-54	0.15	0.004	0.07	0.46	0	0.09	0.2	0	0.04
55-59	0.14	0.004	0.07	0.399	0.0002	0.08	0.29	0	0.04
60-64	0.11	0.0005	0.06	0.38	0	0.08	0.31	0.002	0.06
65-69	0.09	0.0003	0.04	0.32	0	0.08	0.41	0.002	0.07
70-74	0.06	0.002	0.03	0.28	0	0.07	0.45	0	0.1
75-79	0.06	0	0.04	0.26	0	0.1	0.39	0.002	0.14
80-84	0.03	0.001	0.006	0.3	0.003	0.13	0.38	0	0.14
85+	0.003	0.0007	0.02	0.25	0.002	0.21	0.31	0.02	0.199
White Female									
45-49	0.11	0.01	0.05	0.4	0.006	0.24	0.12	0.006	0.06
50-54	0.12	0.004	0.06	0.33	0.008	0.26	0.15	0.002	0.06
55-59	0.13	0.005	0.07	0.37	0.005	0.2	0.16	0.003	0.06
60-64	0.1	0.003	0.07	0.37	0.005	0.19	0.18	0.003	0.08
65-69	0.09	0.008	0.05	0.38	0.008	0.22	0.16	0.003	0.08
70-74	0.06	0.008	0.05	0.39	0.008	0.18	0.21	0.002	0.09
75-79	0.04	0.008	0.04	0.38	0.005	0.18	0.23	0.004	0.11
80-84	0.02	0.003	0.03	0.37	0.02	0.2	0.21	0.008	0.15
85+	0.01	0	0.02	0.37	0.03	0.23	0.2	0.009	0.13
Black Male									
45-49	0.15	0	0.07	0.49	0	0.15	0.1	0	0.05
50-54	0.24	0	0.02	0.52	0	0.07	0.13	0	0.019
55-59	0.12	0	0.1	0.43	0.02	0.14	0.2	0	0.004
60-64	0.17	0	0.08	0.46	0	0.099	0.19	0.002	0.01
65-69	0.12	0	0.11	0.43	0	0.13	0.18	0	0.03
70-74	0.1	0.007	0.12	0.24	0.01	0.08	0.35	0	0.09
75-79	0.03	0.03	0.08	0.28	0	0.06	0.42	0	0.1
80-84	0.05	0.11	0.12	0.23	0	0.15	0.25	0	0.11
85+	0	0	0	0.39	0	0.18	0.2	0	0.23
Black Female									
45-49	0.13	0	0.03	0.54	0	0.26	0.04	0.004	0.002
50-54	0.12	0.01	0.02	0.58	0	0.15	0.11	0	0.007
55-59	0.13	0	0.04	0.58	0.004	0.12	0.12	0	0.006
60-64	0.13	0.007	0.03	0.51	0.004	0.12	0.19	0.001	0.01
65-69	0.08	0	0.1	0.49	0	0.11	0.16	0	0.06
70-74	0.11	0	0.04	0.53	0.005	0.11	0.14	0	0.05
75-79	0.06	0	0.03	0.5	0	0.14	0.24	0	0.03
80-84	0.04	0	0.009	0.5	0	0.15	0.22	0	0.08
85+	0.01	0	0	0.45	0.07	0.06	0.2	0	0.2

There are total of four race-gender groups: white male, white female, black male and black female. Within each race-gender group, there are three possible smoking status (smoker, never-smoker and former smoker) and three possible weight status (underweight, normal-weight, overweight).

Table 2.2. Relative risk (RR) adjusted for smoking and weight statuses by gender.

Relative Risk (RR)	Male	Female
Overweight Smoker	4.025	3.48
Underweight Smoker	3.93	3.25
Normal-weight Smoker	2.8	2.76
Overweight Never-Smoker	2.225	1.72
Underweight Never-Smoker	2.13	1.49
Normal-weight Never-Smoker	1	1
Overweight Former Smoker	2.695	2.17
Underweight Former Smoker	2.6	1.94
Normal-weight Former Smoker	1.47	1.45

For both male and female, there are three possible smoking status (smoker, never-smoker and former smoker) and three possible weight status (underweight, normal-weight, overweight).

Table 2.3. Age-specific annual mortality rates for normal-weight never-smokers by gender and race.

Age	White Male (%)	White Female (%)	Black Male (%)	Black Female (%)
40-44	0.12	0.10	0.18	0.14
45-49	0.16	0.14	0.22	0.19
50-54	0.25	0.20	0.36	0.30
55-59	0.36	0.30	0.53	0.43
60-64	0.53	0.43	0.86	0.62
65-69	0.73	0.63	1.12	0.86
70-74	1.13	1.05	1.57	1.28
75-79	1.86	1.79	2.20	1.99
80-84	3.22	3.19	4.08	3.29
85-99	7.64	8.87	8.23	7.47

a We assume the maximum life span is 100 years.

Table 2.4 Baseline characteristics of the sample population, Health and Retirement Study 1992.

Characteristics	N = 8,958
Age (years)	55.22 ± 5.55
Male (%)	43.03
White Race (%)	80.23
Weight (kg)	77.62 ± 16.62
Body Mass Index (BMI)	26.99 ± 5.03
Normal Weight (BMI < 25 kg/m^2)	36.68
Overweight (25 kg/m^2 ≤ BMI < 30 kg/m^2)	40.39
Obese (BMI ≥ 30 kg/m^2)	22.93
Height (m)	1.69 ± 0.099
Smoking status (%)	
Current Smoker	30.53
Former Smoker	25.11
Never Smoker	44.36
Marital Status (%)	
Married/Partnered	81.16
Single	2.88
Divorced/Widowed	15.95
Education (%)	
Less than High School	30.22
High School Graduate	33.05
Some College	19.41
College and Above	17.31

Table 2.5 Regression results for change in BMI (%) under Model I, Model II and Model III.

Change in BMI (%) between any two consecutive surveys	Never Smokers (N = 33,458) Model I	Never Smokers (N = 33,548) Model II	Never Smokers (N = 35,363) Model III	Current Smokers (N = 13,607) Model I	Current Smokers (N = 14,032) Model II	Current Smokers (N = 15,658) Model III
Age	-0.00063^{***}	-0.00063^{***}	-0.00067^{***}	-0.00057^{***}	-0.00060^{***}	-0.00079^{***}
	(0.00004)	(0.00004)	(0.0000457)	(0.0000766)	(0.0000755)	(0.0000828)
Sex	0.00132^{**}	0.00132^{**}	0.00242^{**}	0.0007	0.00049	0.00217^{*}
	(0.00059)	(0.00059)	(0.0007057)	(0.0010777)	(0.0010603)	(0.0012124)
SES	0.00091	0.00091	−0.00125	0.00007	0.00054	−0.00186
	(0.00075)	(0.00075)	(0.0009541)	(0.00118)	(0.0011566)	(0.0013455)
Race	-0.00108^{*}	-0.00108^{*}	−0.00098	-0.00239^{**}	-0.0023^{**}	-0.0032^{**}
	(0.0006)	(0.0006)	(0.0007043)	(0.00107)	(0.0010553)	(0.0011998)
BMI (previous survey)	-0.00167^{***}	-0.00167^{***}	-0.00236^{***}	-0.00188^{***}	-0.00187^{***}	-0.00281^{***}
	(0.00008)	(0.00008)	(0.0001107)	(0.00014)	(0.0001386)	(0.0002079)

*Note. Standard errors are in parenthesis. * p < 0.10, ** p<0.05, and *** p<0.01.*

Figure 2.1 Flowchart for selecting eligible participants in the final sample from the Health and Retirement Study.

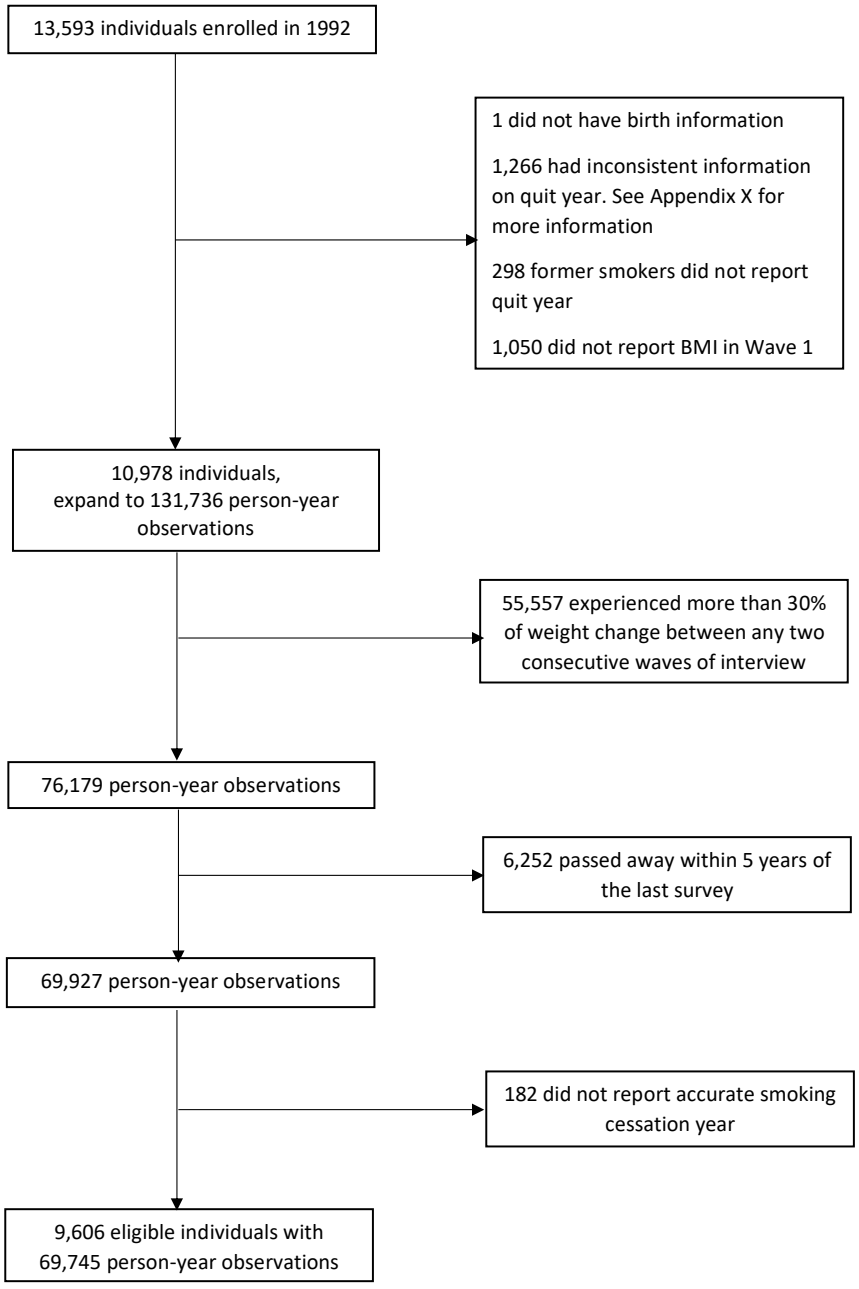
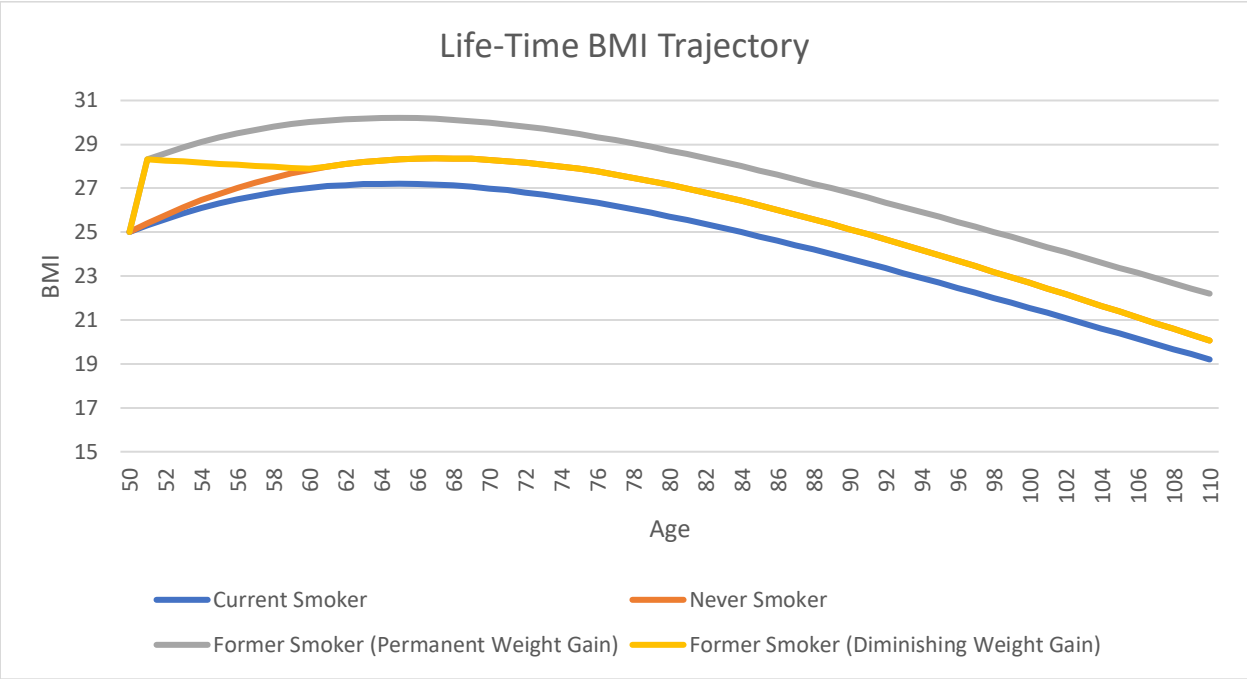
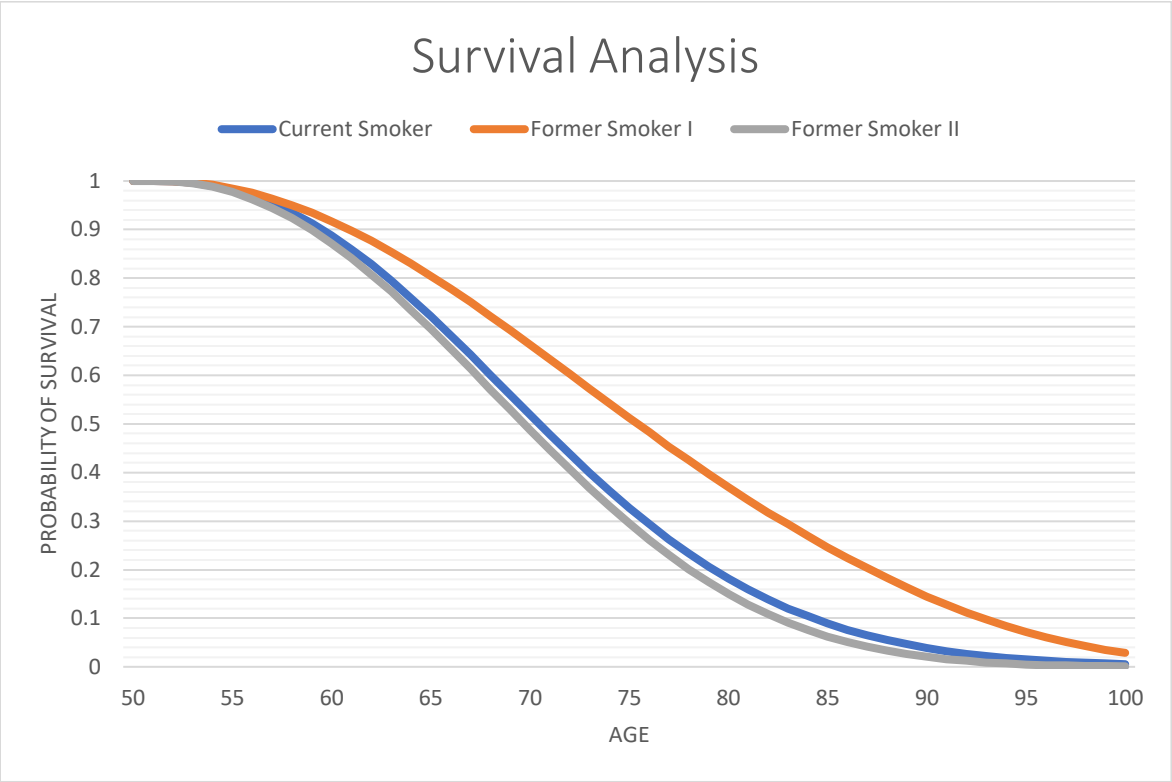


Figure 2.2 Simulated life-time BMI trajectory for a white male with an initial BMI of 25 at age 50, assuming different smoking behaviors.



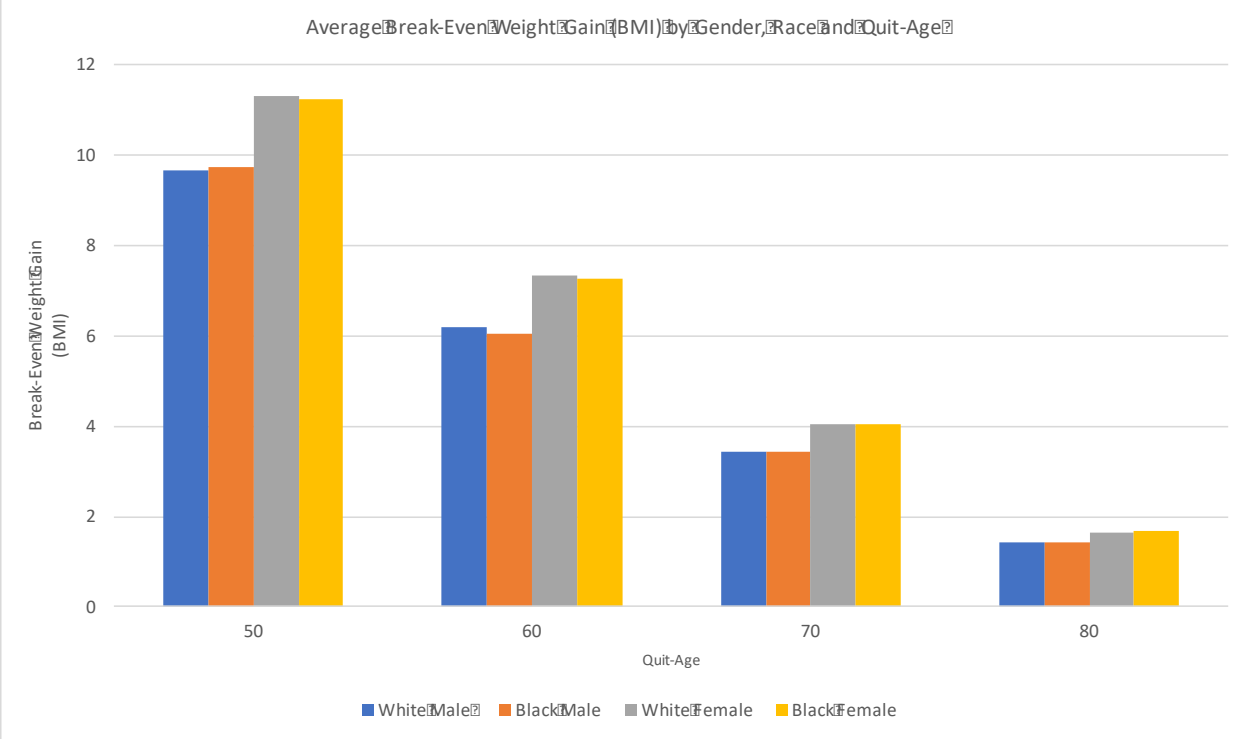
Note. Blue curve = current smoker; orange curve = never smoker; grey curve = former smoker with permanent weight gain post cessation; yellow curve = former smoker with diminishing weight gain post cessation.

Figure 2.3 Survival curves for current smoker vs former smoker I vs former smoker II from age 50 to 100.



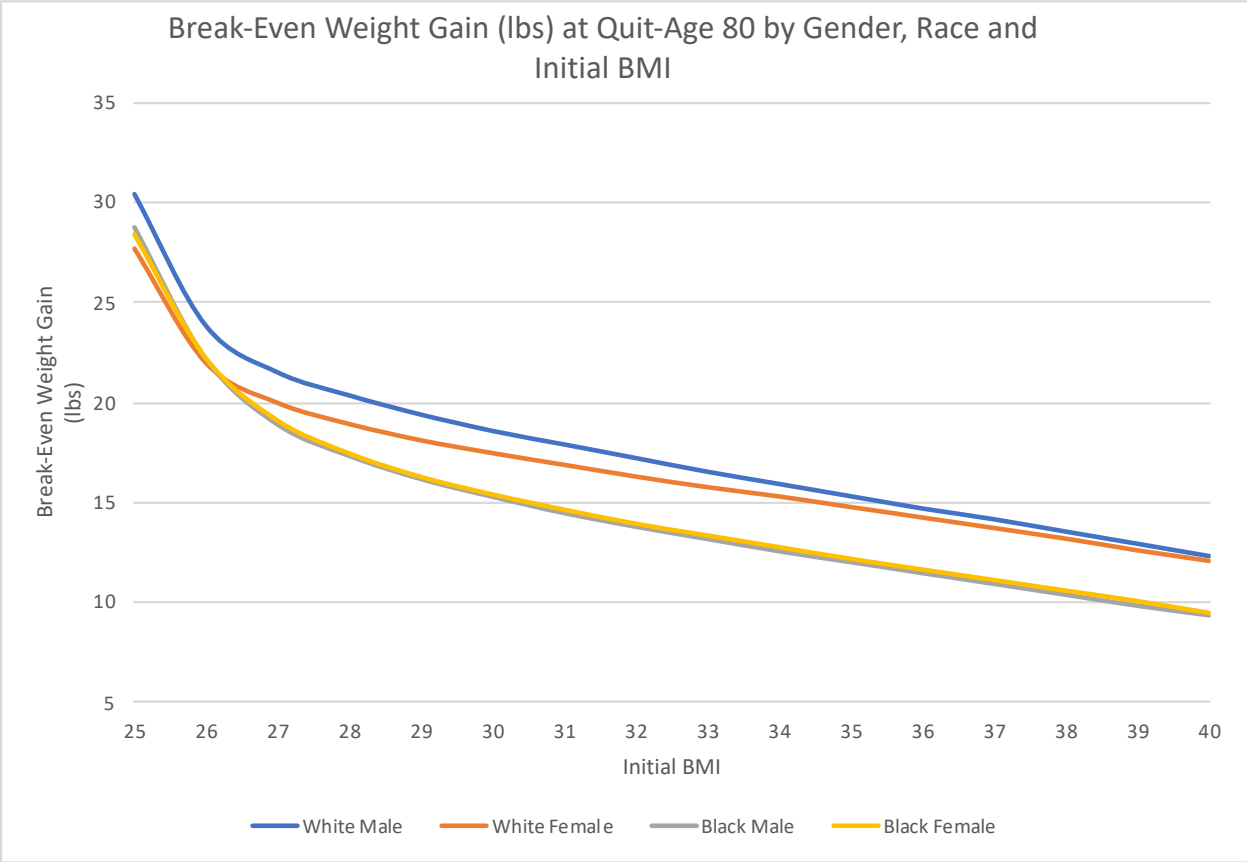
Note. Blue curve = current smokers; orange curve = former smoker I with no post-cessation weight gain; grey curve = former smoker II with substantial amount of post-cessation weight gain.

Figure 2.4 Average break-even weight gain (BMI) by gender, race and quit-age under permanent weight gain.



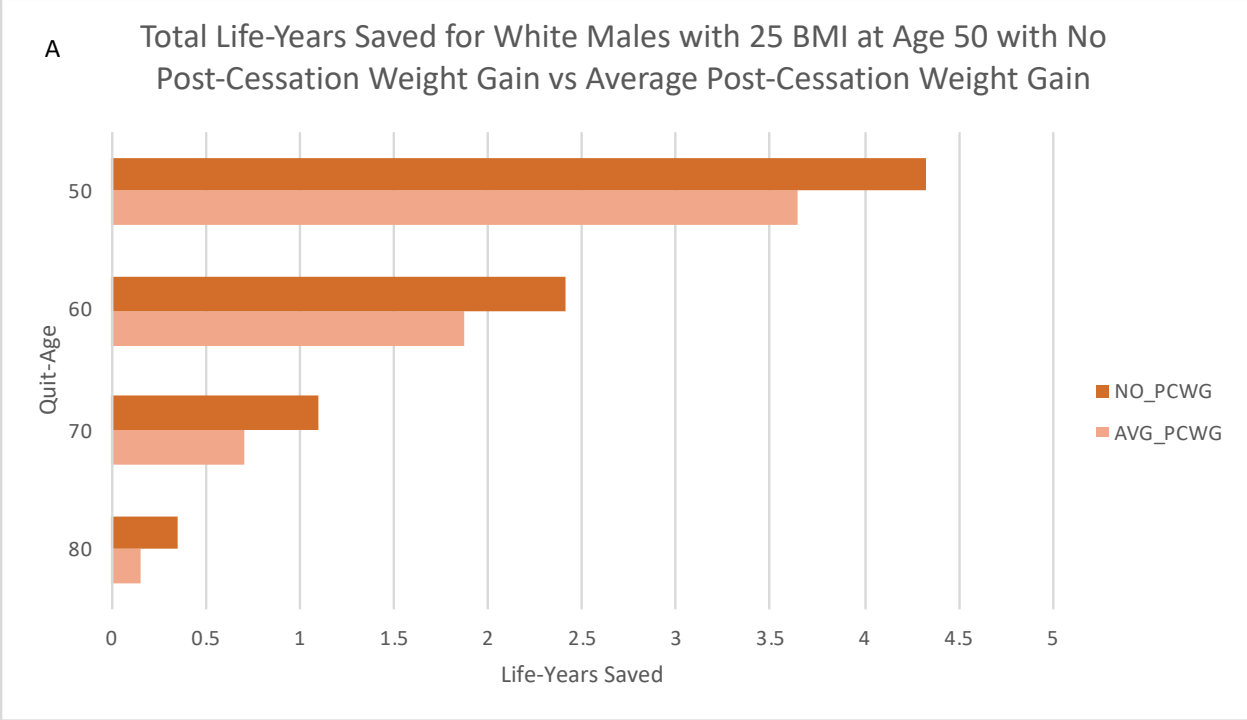
Note. Blue column = white males; orange column = black males; grey column = white females; yellow column = black females.

Figure 2.5 Break-even weight gain (lbs) at quit-age 80 by gender, race and initial BMI under diminishing weight-gain.

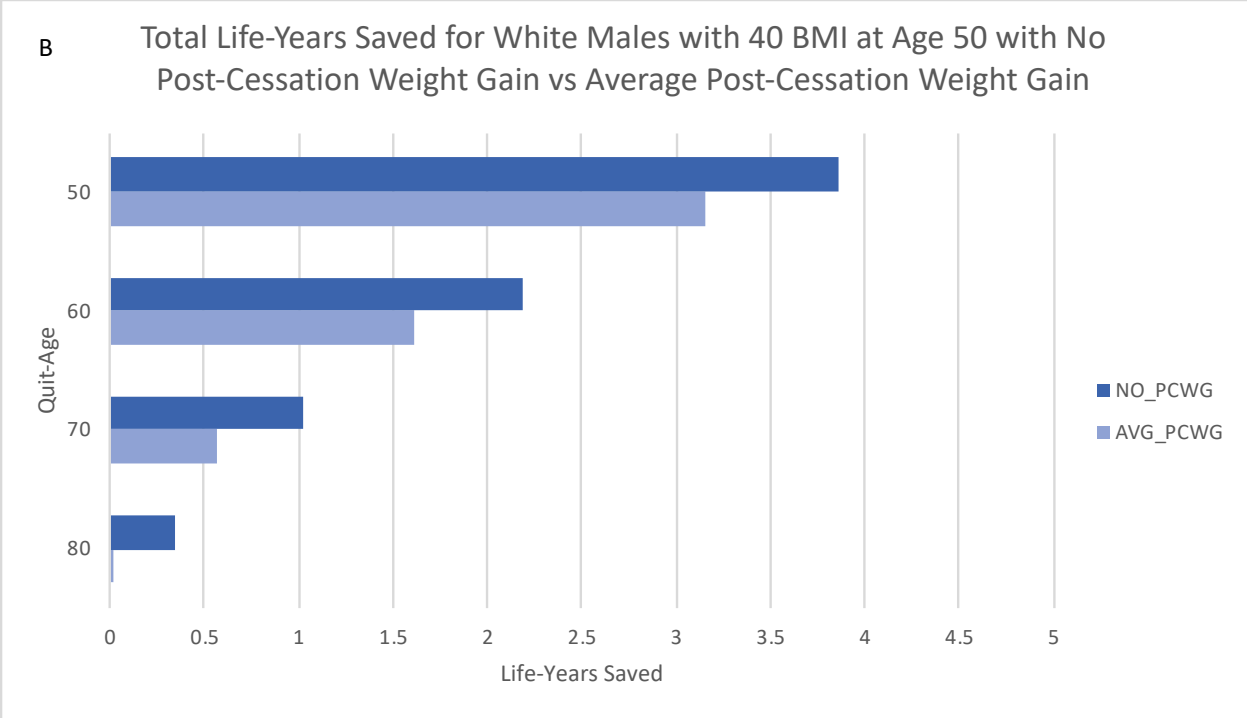


Note. Blue curve = white males; orange curve = white females; grey curve = black males; yellow curve = black females.

Figure 2.6 Total life-years saved for white males with no post-cessation weight gain vs average post-cessation weight gain with known initial BMI at age 50.



Note. Dark orange bar = white males with an initial BMI of 25 at age 50 and no weight gain after quitting; light orange bar = white males with an initial BMI of 25 at age 50 and a post-cessation weight gain of 10.3 lbs.



Note. Dark blue bar = white males with an initial BMI of 40 at age 50 and no weight gain after quitting; light blue bar = white males with an initial BMI of 40 at age 50 and a post-cessation weight gain of 10.3 lbs.

Appendices

Appendix 2.A Break-even weight gain in BMI units by initial BMI, quit-age, race and gender under permanent weight gain with high SES.

Table 2.A.1 Break-even weight gain in BMI units for males with high SES under permanent weight gain.

	White Male				Black Male			
Initial BMI/ Quit-Age	50	60	70	80	50	60	70	80
25	10.88	6.31	3.69	2.38	10.12	6.43	4.18	2.62
26	10.88	6.25	3.42	1.87	10.08	6.31	3.80	2.06
27	10.87	6.21	3.30	1.62	10.05	6.23	3.61	1.78
28	10.86	6.18	3.23	1.49	10.03	6.16	3.48	1.61
29	10.86	6.15	3.17	1.41	10.01	6.12	3.39	1.51
30	10.86	6.13	3.14	1.36	10.00	6.08	3.33	1.44
31	10.86	6.11	3.11	1.32	9.98	6.05	3.27	1.39
32	10.86	6.10	3.08	1.29	9.97	6.02	3.23	1.35
33	10.86	6.09	3.06	1.27	9.96	5.99	3.20	1.32
34	10.86	6.07	3.04	1.25	9.95	5.97	3.17	1.30
35	10.86	6.06	3.03	1.23	9.94	5.95	3.14	1.28
36	10.86	6.05	3.01	1.22	9.93	5.94	3.12	1.26
37	10.86	6.04	3.00	1.21	9.92	5.92	3.10	1.25
38	10.86	6.03	2.99	1.20	9.92	5.91	3.08	1.24
39	10.86	6.03	2.98	1.19	9.91	5.89	3.07	1.23
40	10.86	6.02	2.97	1.19	9.90	5.88	3.05	1.22

Table 2.A.2 Break-even weight gain in BMI units for females with high SES under permanent weight gain.

	White Female				Black Female			
Initial BMI/ Quit-Age	50	60	70	80	50	60	70	80
25	12.98	7.93	4.21	2.23	12.65	7.96	4.68	2.55
26	12.98	7.87	3.99	1.75	12.61	7.83	4.31	2.00
27	12.97	7.84	3.89	1.57	12.58	7.75	4.13	1.74
28	12.97	7.82	3.83	1.48	12.56	7.69	4.01	1.60
29	12.97	7.80	3.79	1.43	12.54	7.64	3.93	1.51
30	12.96	7.78	3.76	1.40	12.53	7.60	3.87	1.45
31	12.96	7.77	3.74	1.38	12.51	7.58	3.83	1.42
32	12.96	7.77	3.72	1.37	12.50	7.55	3.80	1.39
33	12.97	7.76	3.71	1.36	12.50	7.53	3.77	1.37
34	12.97	7.75	3.70	1.36	12.49	7.52	3.75	1.36
35	12.97	7.75	3.69	1.35	12.48	7.50	3.73	1.35
36	12.97	7.74	3.68	1.35	12.48	7.49	3.72	1.34
37	12.97	7.74	3.68	1.34	12.47	7.48	3.70	1.33
38	12.97	7.74	3.67	1.34	12.47	7.47	3.69	1.33
39	12.97	7.73	3.66	1.34	12.47	7.47	3.68	1.32
40	12.98	7.73	3.66	1.34	12.46	7.46	3.67	1.32

Appendix 2.B Break-even weight gain in BMI units for smokers with low SES under permanent weight gain.

Table 2.B.1 Break-even weight gain in BMI units for males with low SES under permanent weight gain.

	White Male				Black Male			
Initial BMI/Quit-Age	50	60	70	80	50	60	70	80
25	10.1	6.6	4.0	2.3	10.1	6.7	4.3	2.5
26	10.1	6.5	3.7	1.8	10.0	6.5	3.9	1.9
27	10.1	6.4	3.6	1.6	10.0	6.4	3.6	1.6
28	10.0	6.4	3.5	1.5	9.9	6.3	3.5	1.5
29	10.0	6.4	3.4	1.4	9.9	6.3	3.4	1.4
30	10.0	6.3	3.4	1.4	9.9	6.3	3.4	1.4
31	10.0	6.3	3.3	1.3	9.9	6.2	3.3	1.3
32	10.0	6.3	3.3	1.3	9.9	6.2	3.3	1.3
33	10.0	6.3	3.3	1.3	9.9	6.2	3.3	1.3
34	10.0	6.3	3.3	1.3	9.9	6.1	3.2	1.3
35	10.0	6.3	3.3	1.3	9.8	6.1	3.2	1.3
36	10.0	6.3	3.3	1.3	9.8	6.1	3.2	1.2
37	10.0	6.3	3.3	1.3	9.8	6.1	3.2	1.2
38	10.0	6.3	3.2	1.2	9.8	6.1	3.2	1.2
39	10.0	6.2	3.2	1.2	9.8	6.1	3.2	1.2
40	10.0	6.2	3.2	1.2	9.8	6.1	3.1	1.2

Table 2.B.2 Break-even weight gain in BMI units for females with low SES under permanent weight gain.

	White Female				Black Female			
Initial BMI/Quit-Age	50	60	70	80	50	60	70	80
25	11.6	7.7	4.6	2.4	11.5	7.9	4.9	2.6
26	11.6	7.6	4.3	1.9	11.5	7.7	4.5	2.0
27	11.6	7.6	4.2	1.7	11.5	7.6	4.3	1.8
28	11.6	7.5	4.1	1.6	11.4	7.6	4.1	1.6
29	11.6	7.5	4.0	1.5	11.4	7.5	4.1	1.6
30	11.6	7.5	4.0	1.5	11.4	7.5	4.0	1.5
31	11.6	7.5	4.0	1.5	11.4	7.5	4.0	1.5
32	11.6	7.5	4.0	1.5	11.4	7.4	3.9	1.5
33	11.6	7.5	4.0	1.5	11.3	7.4	3.9	1.5
34	11.6	7.5	3.9	1.5	11.3	7.4	3.9	1.4
35	11.6	7.4	3.9	1.5	11.3	7.4	3.9	1.4
36	11.6	7.4	3.9	1.5	11.3	7.4	3.9	1.4
37	11.6	7.4	3.9	1.5	11.3	7.4	3.9	1.4
38	11.6	7.4	3.9	1.5	11.3	7.4	3.8	1.4
39	11.6	7.4	3.9	1.5	11.3	7.3	3.8	1.4
40	11.5	7.4	3.9	1.5	11.3	7.3	3.8	1.4

Appendix 2.C Break-even weight gain in BMI units for smokers under diminishing weight gain.

Table 2.C.1Break-even weight gain in BMI units for smokers with low SES under diminishing weight gain.

	White Male		White Female		Black Male		Black Female	
Initial BMI/Quit-Age	70	80	70	80	70	80	70	80
25	14.42	4.30	17.52	4.51	12.18	4.34	16.36	4.55
26	13.87	3.48	16.79	3.69	11.40	3.43	15.37	3.63
27	13.49	3.20	16.30	3.42	10.94	3.04	14.72	3.24
28	13.14	3.08	15.90	3.27	10.57	2.84	14.20	3.03
29	12.83	2.97	15.56	3.16	10.23	2.69	13.79	2.86
30	12.53	2.87	15.24	3.05	9.93	2.55	13.42	2.71
31	12.24	2.77	14.95	2.97	9.66	2.43	13.09	2.59
32	11.97	2.68	14.67	2.88	9.41	2.33	12.79	2.49
33	11.71	2.59	14.41	2.80	9.16	2.23	12.51	2.39
34	11.45	2.51	14.16	2.73	8.93	2.13	12.25	2.30
35	11.20	2.42	13.92	2.65	8.72	2.05	12.00	2.22
36	10.96	2.34	13.68	2.57	8.50	1.96	11.76	2.14
37	10.72	2.26	13.45	2.49	8.30	1.88	11.52	2.05
38	10.49	2.17	13.22	2.41	8.10	1.80	11.29	1.97
39	10.27	2.09	13.00	2.33	7.90	1.71	11.07	1.89
40	10.05	2.01	12.77	2.25	7.71	1.63	10.86	1.80

Appendix 2.D Break-even weight gain calculated via alternative lifetime weight trajectories (Model II and Model III).

Table 2.D.1 Break-even weight gain in BMI units for males with high SES under permanent weight gain (Model II).

	White Male				Black Male			
Initial BMI/ Quit-Age	50	60	70	80	50	60	70	80
25	9.73	6.45	4.12	2.38	9.88	6.47	4.36	2.53
26	9.71	6.37	3.81	1.83	9.84	6.34	3.96	1.94
27	9.69	6.3	3.65	1.58	9.81	6.25	3.74	1.64
28	9.67	6.25	3.53	1.47	9.78	6.18	3.59	1.5
29	9.66	6.21	3.46	1.42	9.76	6.13	3.49	1.42
30	9.65	6.19	3.42	1.38	9.74	6.08	3.41	1.37
31	9.64	6.17	3.38	1.36	9.73	6.05	3.36	1.34
32	9.64	6.15	3.36	1.34	9.71	6.02	3.32	1.32
33	9.63	6.14	3.34	1.33	9.7	5.99	3.29	1.3
34	9.62	6.13	3.33	1.33	9.69	5.97	3.27	1.29
35	9.62	6.12	3.32	1.32	9.68	5.96	3.25	1.28
36	9.62	6.11	3.31	1.32	9.67	5.94	3.24	1.28
37	9.61	6.1	3.3	1.31	9.66	5.93	3.23	1.27
38	9.61	6.1	3.3	1.31	9.66	5.92	3.21	1.27
39	9.61	6.09	3.29	1.31	9.65	5.91	3.21	1.26
40	9.61	6.09	3.28	1.31	9.65	5.9	3.2	1.26

Table 2.D.2 Break-even weight gain in BMI units for females with high SES under permanent weight gain (Model II).

	White Female				Black Female			
Initial BMI/Quit-Age	50	60	70	80	50	60	70	80
25	11.36	7.59	4.64	2.51	11.41	7.69	4.97	2.77
26	11.34	7.51	4.36	1.96	11.37	7.56	4.59	2.19
27	11.33	7.45	4.2	1.74	11.34	7.47	4.37	1.88
28	11.31	7.41	4.1	1.65	11.31	7.4	4.22	1.72
29	11.3	7.38	4.04	1.6	11.29	7.34	4.11	1.64
30	11.3	7.36	4.01	1.57	11.27	7.3	4.04	1.59
31	11.29	7.34	3.98	1.56	11.25	7.26	3.99	1.56
32	11.29	7.33	3.97	1.55	11.24	7.23	3.96	1.54
33	11.28	7.32	3.95	1.54	11.23	7.21	3.93	1.53
34	11.28	7.31	3.94	1.53	11.22	7.19	3.91	1.52
35	11.27	7.3	3.94	1.53	11.21	7.17	3.89	1.51
36	11.27	7.3	3.93	1.53	11.2	7.16	3.88	1.51
37	11.27	7.29	3.93	1.53	11.19	7.15	3.87	1.51
38	11.27	7.29	3.92	1.52	11.18	7.14	3.86	1.5
39	11.26	7.29	3.92	1.52	11.18	7.13	3.85	1.5
40	11.26	7.28	3.91	1.52	11.17	7.12	3.85	1.5

Table 2.D.3 Break-even weight gain in BMI units for males with low SES under permanent weight gain (Model II).

	White Male				Black Male			
Initial BMI/ Quit-Age	50	60	70	80	50	60	70	80
25	9.77	6.53	4.23	2.44	9.93	6.56	4.46	2.59
26	9.75	6.43	3.89	1.88	9.89	6.41	4.04	1.99
27	9.72	6.35	3.71	1.62	9.85	6.31	3.8	1.68
28	9.7	6.3	3.58	1.49	9.82	6.23	3.65	1.52
29	9.69	6.25	3.5	1.43	9.8	6.17	3.53	1.43
30	9.67	6.22	3.44	1.39	9.78	6.12	3.45	1.38
31	9.66	6.19	3.4	1.37	9.76	6.08	3.39	1.35
32	9.65	6.17	3.38	1.35	9.74	6.05	3.35	1.33
33	9.65	6.16	3.36	1.34	9.73	6.02	3.31	1.31
34	9.64	6.14	3.34	1.33	9.72	6	3.29	1.3
35	9.63	6.13	3.33	1.32	9.7	5.98	3.27	1.29
36	9.63	6.12	3.32	1.32	9.69	5.96	3.25	1.28
37	9.62	6.11	3.31	1.32	9.68	5.95	3.24	1.27
38	9.62	6.11	3.3	1.31	9.68	5.93	3.22	1.27
39	9.62	6.1	3.29	1.31	9.67	5.92	3.21	1.26
40	9.61	6.1	3.29	1.31	9.66	5.91	3.2	1.26

Table 2.D.4 Break-even weight gain in BMI units for females with low SES under permanent weight gain (Model II).

	White Female				Black Female			
Initial BMI/Quit-Age	50	60	70	80	50	60	70	80
25	11.41	7.67	4.75	2.57	11.47	7.79	5.08	2.84
26	11.38	7.57	4.44	2.02	11.42	7.64	4.67	2.25
27	11.36	7.5	4.26	1.77	11.39	7.53	4.43	1.91
28	11.34	7.45	4.14	1.66	11.36	7.45	4.28	1.75
29	11.33	7.41	4.07	1.61	11.33	7.39	4.16	1.66
30	11.32	7.38	4.03	1.58	11.31	7.34	4.08	1.61
31	11.31	7.36	4	1.56	11.29	7.3	4.02	1.57
32	11.3	7.34	3.98	1.55	11.27	7.27	3.98	1.55
33	11.29	7.33	3.96	1.54	11.26	7.24	3.95	1.54
34	11.29	7.32	3.95	1.54	11.24	7.22	3.93	1.53
35	11.28	7.31	3.94	1.53	11.23	7.2	3.91	1.52
36	11.28	7.31	3.94	1.53	11.22	7.18	3.89	1.51
37	11.28	7.3	3.93	1.53	11.21	7.17	3.88	1.51
38	11.27	7.3	3.93	1.52	11.2	7.16	3.87	1.5
39	11.27	7.29	3.92	1.52	11.2	7.15	3.86	1.5
40	11.27	7.29	3.92	1.52	11.19	7.14	3.85	1.5

Table 2.D.5 Break-even weight gain in BMI units for males with high SES under permanent weight gain (Model III).

	White Male				Black Male			
Initial BMI/ Quit-Age	50	60	70	80	50	60	70	80
25	10.16	7.07	4.89	2.98	10.37	7.19	5.18	3.17
26	10.13	6.96	4.54	2.42	10.33	7.01	4.78	2.56
27	10.11	6.87	4.33	2.05	10.29	6.89	4.52	2.17
28	10.09	6.81	4.17	1.83	10.26	6.8	4.32	1.91
29	10.07	6.75	4.05	1.7	10.23	6.73	4.17	1.74
30	10.05	6.7	3.95	1.61	10.21	6.67	4.05	1.64
31	10.04	6.66	3.87	1.55	10.19	6.62	3.95	1.56
32	10.02	6.63	3.8	1.5	10.17	6.58	3.87	1.51
33	10.01	6.6	3.74	1.47	10.15	6.53	3.8	1.47
34	10	6.57	3.69	1.44	10.13	6.5	3.74	1.44
35	9.99	6.54	3.65	1.42	10.12	6.47	3.69	1.41
36	9.98	6.52	3.61	1.41	10.11	6.44	3.65	1.39
37	9.97	6.5	3.58	1.39	10.1	6.42	3.6	1.37
38	9.96	6.48	3.56	1.38	10.09	6.39	3.57	1.36
39	9.95	6.47	3.54	1.37	10.07	6.37	3.54	1.35
40	9.95	6.45	3.52	1.36	10.06	6.35	3.51	1.34

Table 2.D.6 Break-even weight gain in BMI units for females with high SES under permanent weight gain (Model III).

	White Female				Black Female			
Initial BMI/Quit-Age	50	60	70	80	50	60	70	80
25	11.64	7.96	5.14	2.94	11.78	8.18	5.56	3.29
26	11.62	7.88	4.84	2.42	11.74	8.05	5.19	2.72
27	11.6	7.82	4.66	2.08	11.71	7.95	4.96	2.34
28	11.59	7.77	4.53	1.89	11.69	7.88	4.79	2.08
29	11.58	7.73	4.43	1.79	11.67	7.82	4.65	1.93
30	11.56	7.7	4.35	1.72	11.65	7.77	4.55	1.83
31	11.55	7.66	4.28	1.68	11.63	7.72	4.47	1.76
32	11.54	7.64	4.23	1.64	11.62	7.68	4.4	1.71
33	11.53	7.61	4.19	1.62	11.6	7.65	4.33	1.68
34	11.53	7.59	4.16	1.6	11.59	7.62	4.28	1.64
35	11.52	7.58	4.13	1.59	11.58	7.59	4.23	1.62
36	11.51	7.56	4.11	1.58	11.57	7.57	4.2	1.6
37	11.5	7.54	4.09	1.57	11.56	7.55	4.16	1.59
38	11.5	7.53	4.07	1.56	11.55	7.53	4.14	1.58
39	11.49	7.51	4.06	1.56	11.54	7.51	4.11	1.57
40	11.49	7.5	4.04	1.55	11.53	7.5	4.09	1.56

Table 2.D.7 Break-even weight gain in BMI units for males with low SES under permanent weight gain (Model III).

	White Male				Black Male			
Initial BMI/ Quit-Age	50	60	70	80	50	60	70	80
25	9.97	6.78	4.57	2.78	10.16	6.87	4.87	2.97
26	9.95	6.71	4.26	2.25	10.13	6.74	4.49	2.39
27	9.94	6.64	4.08	1.91	10.1	6.65	4.26	2.03
28	9.92	6.59	3.95	1.72	10.08	6.58	4.09	1.79
29	9.91	6.55	3.85	1.61	10.06	6.52	3.96	1.66
30	9.89	6.51	3.77	1.54	10.04	6.48	3.86	1.57
31	9.88	6.48	3.7	1.49	10.02	6.44	3.78	1.51
32	9.87	6.46	3.65	1.46	10.01	6.4	3.71	1.46
33	9.87	6.44	3.6	1.43	10	6.37	3.66	1.43
34	9.86	6.41	3.57	1.41	9.99	6.34	3.61	1.4
35	9.85	6.39	3.54	1.39	9.98	6.31	3.56	1.38
36	9.84	6.38	3.51	1.38	9.97	6.29	3.53	1.36
37	9.84	6.36	3.49	1.37	9.96	6.27	3.5	1.35
38	9.83	6.35	3.47	1.36	9.95	6.25	3.47	1.34
39	9.83	6.33	3.46	1.35	9.94	6.24	3.45	1.33
40	9.82	6.32	3.44	1.35	9.93	6.22	3.43	1.32

Table 2.D.8 Break-even weight gain in BMI units for females with low SES under permanent weight gain (Model III).

	White Female				Black Female			
Initial BMI/Quit-Age	50	60	70	80	50	60	70	80
25	11.49	7.72	4.81	2.73	11.57	7.88	5.23	3.07
26	11.47	7.67	4.58	2.22	11.55	7.78	4.9	2.53
27	11.46	7.63	4.44	1.94	11.53	7.71	4.7	2.17
28	11.45	7.59	4.34	1.8	11.51	7.66	4.56	1.97
29	11.44	7.56	4.26	1.72	11.5	7.61	4.45	1.84
30	11.43	7.54	4.2	1.67	11.48	7.57	4.37	1.76
31	11.43	7.51	4.15	1.63	11.47	7.54	4.3	1.7
32	11.42	7.49	4.11	1.61	11.46	7.51	4.24	1.66
33	11.41	7.48	4.09	1.59	11.45	7.49	4.19	1.63
34	11.41	7.46	4.07	1.58	11.44	7.46	4.15	1.61
35	11.4	7.45	4.05	1.57	11.43	7.44	4.12	1.59
36	11.4	7.44	4.03	1.56	11.42	7.43	4.09	1.58
37	11.39	7.43	4.02	1.55	11.42	7.41	4.06	1.56
38	11.39	7.42	4.01	1.55	11.41	7.39	4.04	1.56
39	11.38	7.41	4	1.54	11.4	7.38	4.03	1.55
40	11.38	7.4	3.99	1.54	11.4	7.37	4.01	1.54

Table 2.D.9 Break-even weight gain in BMI units for smokers with high SES under diminishing weight gain (Model II).

	White Male		White Female		Black Male		Black Female	
Initial BMI/Quit-Age	70	80	70	80	70	80	70	80
25	14.98	4.48	17.77	4.75	12.78	4.25	16.25	4.87
26	14.19	3.48	16.72	3.71	11.89	3.26	15.07	3.79
27	13.6	3.1	15.93	3.31	11.26	2.76	14.25	3.22
28	13.04	2.9	15.32	3.09	10.73	2.51	13.59	2.9
29	12.6	2.75	14.85	2.93	10.25	2.32	13.01	2.68
30	12.23	2.62	14.47	2.81	9.84	2.18	12.55	2.5
31	11.89	2.51	14.14	2.7	9.51	2.06	12.17	2.36
32	11.59	2.41	13.83	2.6	9.2	1.96	11.84	2.24
33	11.3	2.31	13.55	2.51	8.92	1.86	11.54	2.13
34	11.02	2.22	13.28	2.42	8.67	1.77	11.26	2.03
35	10.76	2.13	13.02	2.33	8.43	1.69	11	1.93
36	10.51	2.04	12.77	2.24	8.2	1.61	10.76	1.84
37	10.27	1.95	12.52	2.15	7.99	1.53	10.52	1.75
38	10.03	1.87	12.29	2.06	7.78	1.46	10.3	1.66
39	9.8	1.78	12.05	1.97	7.58	1.38	10.08	1.57
40	9.57	1.69	11.83	1.88	7.38	1.3	9.86	1.47

Table 2.D.10 Break-even weight gain in BMI units for smokers with low SES under diminishing weight gain (Model II).

	White Male		White Female		Black Male		Black Female	
Initial BMI/Quit-Age	70	80	70	80	70	80	70	80
25	15.18	4.62	18.12	4.92	74.28	28.98	12.96	4.37
26	14.48	3.63	17.2	3.87	73.29	28.2	12.12	3.39
27	14	3.21	16.47	3.44	72.35	27.57	11.58	2.87
28	13.49	3.02	15.84	3.22	71.48	26.98	11.09	2.62
29	13.06	2.88	15.37	3.07	70.66	26.44	10.64	2.44
30	12.68	2.75	14.98	2.94	69.87	25.94	10.23	2.3
31	12.35	2.64	14.63	2.83	69.13	25.47	9.89	2.18
32	12.04	2.54	14.32	2.73	68.43	25.03	9.58	2.08
33	11.75	2.44	14.03	2.64	67.75	24.6	9.3	1.98
34	11.47	2.35	13.75	2.55	67.09	24.2	9.04	1.89
35	11.21	2.26	13.49	2.46	66.47	23.83	8.8	1.81
36	10.95	2.17	13.23	2.37	65.87	23.47	8.57	1.72
37	10.71	2.08	12.99	2.28	65.28	23.12	8.34	1.64
38	10.47	1.99	12.75	2.19	64.72	22.79	8.13	1.56
39	10.23	1.9	12.51	2.09	64.18	22.47	7.92	1.49
40	10.01	1.81	12.28	2	63.65	22.16	7.72	1.41

Table 2.D.11 Break-even weight gain in BMI units for smokers with high SES under diminishing weight gain (Model III).

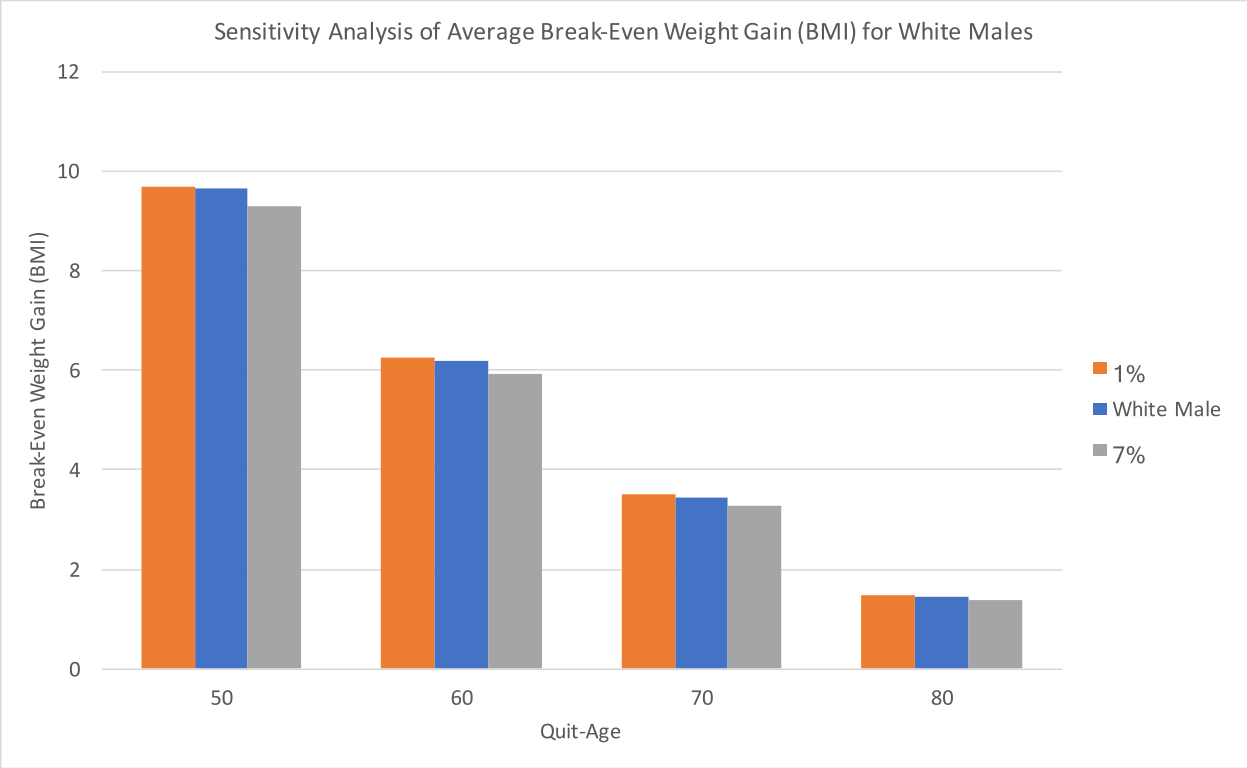
	White Male		White Female		Black Male		Black Female	
Initial BMI/Quit-Age	70	80	70	80	70	80	70	80
25	14.74	4.47	16.5	4.49	12.75	4.3	15.47	4.69
26	13.25	3.33	14.89	3.33	11.36	3.16	13.76	3.48
27	12.17	2.52	13.77	2.49	10.31	2.37	12.56	2.6
28	11.25	1.93	12.89	1.93	9.41	1.76	11.61	1.9
29	10.47	1.48	12.13	1.53	8.64	1.28	10.79	1.4
30	9.78	1.13	11.46	1.22	7.97	0.9	10.11	1
31	9.16	0.82	10.84	0.96	7.38	0.6	9.49	0.67
32	8.58	0.56	10.31	0.73	6.85	0.32	8.93	0.39
33	8.05	0.33	9.82	0.53	6.37	0.09	8.4	0.14
34	7.56	0.12	9.38	0.33	5.91	0	7.92	0
35	7.11	0	8.97	0.15	5.49	0	7.46	0
36	6.68	0	8.58	0	5.1	0	7.05	0
37	6.3	0	8.21	0	4.72	0	6.66	0
38	5.92	0	7.86	0	4.36	0	6.29	0
39	5.56	0	7.52	0	4.03	0	5.94	0
40	5.22	0	7.19	0	3.71	0	5.58	0

Table 2.D.12 Break-even weight gain in BMI units for smokers with low SES under diminishing weight gain (Model III).

	White Male		White Female		Black Male		Black Female	
Initial BMI/Quit-Age	70	80	70	80	70	80	70	80
25	14.06	4.21	15.44	4.21	12.24	4.06	14.57	4.4
26	12.58	3.1	14.08	3.05	10.77	2.95	12.95	3.22
27	11.54	2.33	13.12	2.28	9.74	2.18	11.86	2.34
28	10.71	1.78	12.35	1.8	8.89	1.6	10.99	1.73
29	9.99	1.39	11.67	1.47	8.17	1.16	10.27	1.27
30	9.36	1.07	11.09	1.21	7.57	0.82	9.65	0.92
31	8.78	0.8	10.57	0.98	7.02	0.98	9.09	0.63
32	8.26	0.57	10.12	0.78	6.54	0.54	8.57	0.38
33	7.79	0.36	9.71	0.59	6.09	0.3	8.1	0.14
34	7.36	0.16	9.31	0.42	5.67	0.09	7.67	0
35	6.96	0	8.95	0.25	5.28	0	7.27	0
36	6.58	0	8.6	0.08	4.92	0	6.91	0
37	6.23	0	8.26	0	4.58	0	6.54	0
38	5.88	0	7.93	0	4.26	0	6.21	0
39	5.55	0	7.61	0	3.95	0	5.89	0
40	5.23	0	7.3	0	3.65	0	5.57	0

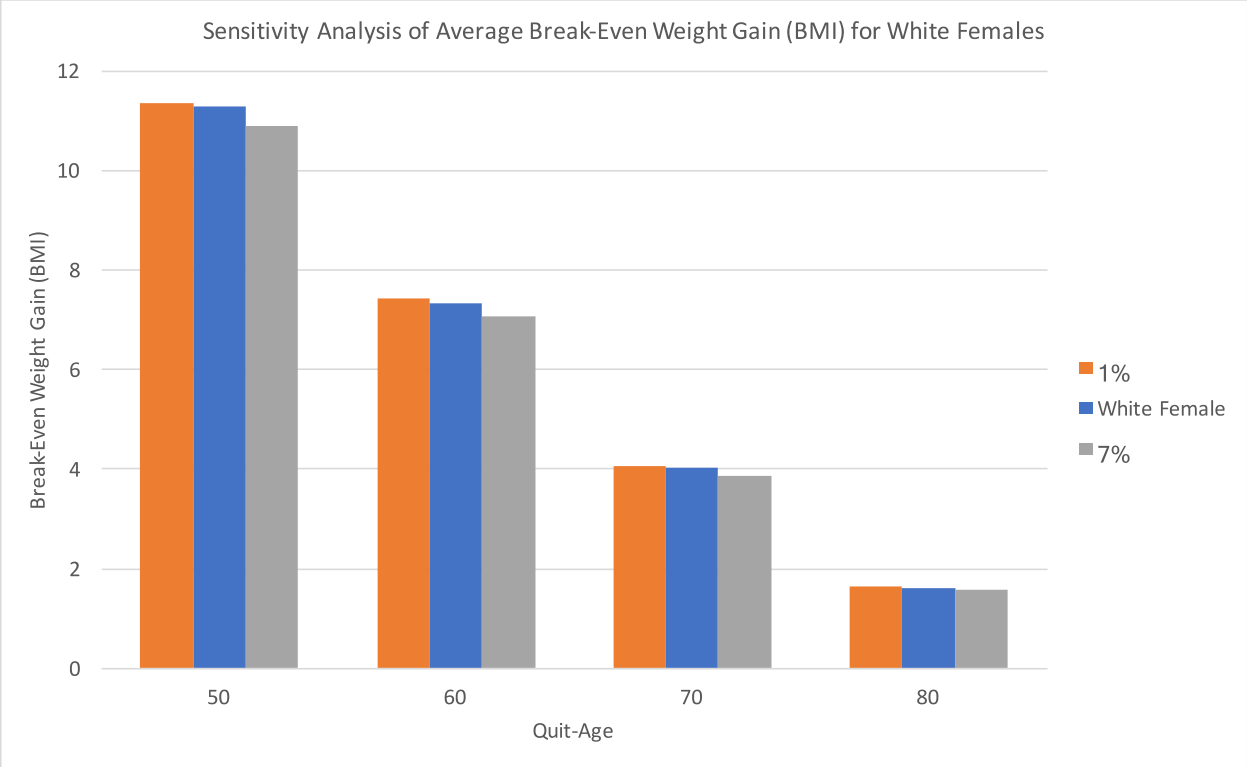
Appendix 2.E Sensitivity analysis with varying discounting rates.

Figure 2.E.1 Sensitivity analysis of average break-even weight gain (BMI) for white males under permanent weight gain.



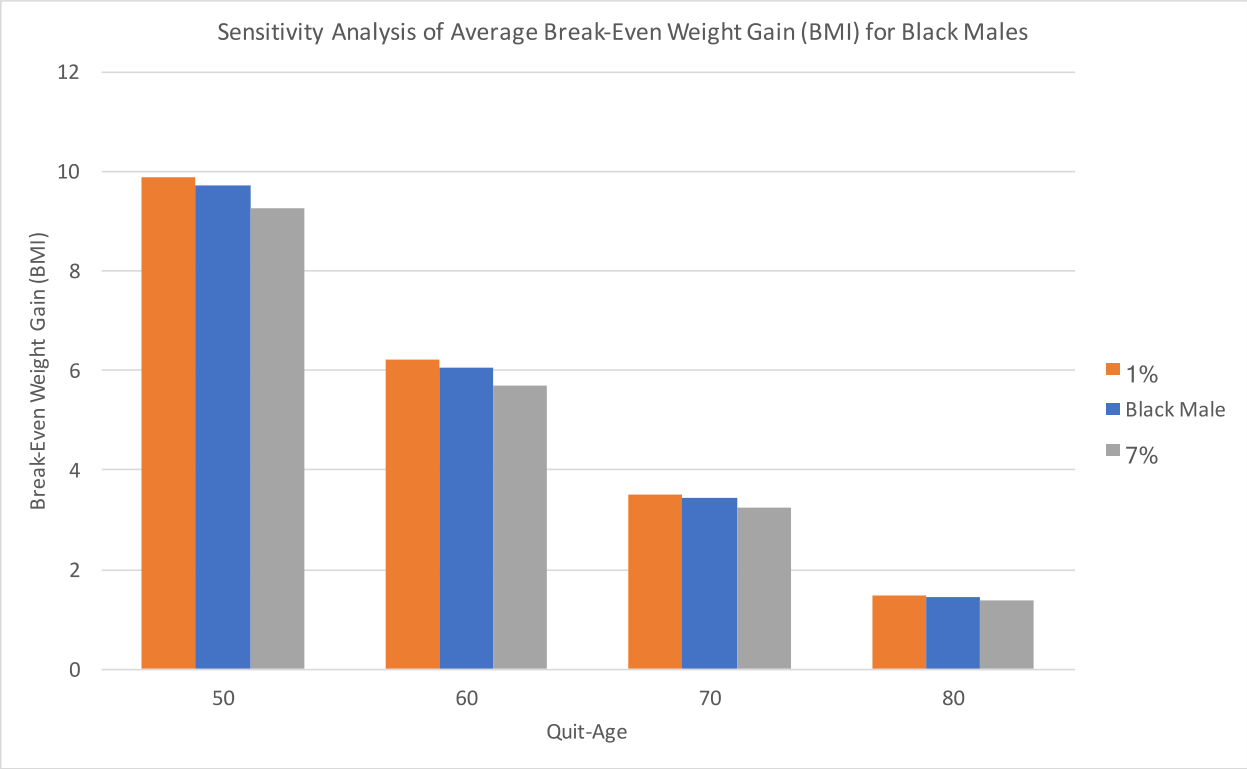
Note. Orange bar = average break-even weight gain in units of BMI for white males with 1% discounting rate; blue bar = 3% discounting rate; grey bar = 7% discounting rate.

Figure 2.E.2 Sensitivity analysis of average break-even weight gain (BMI) for white females under permanent weight gain.



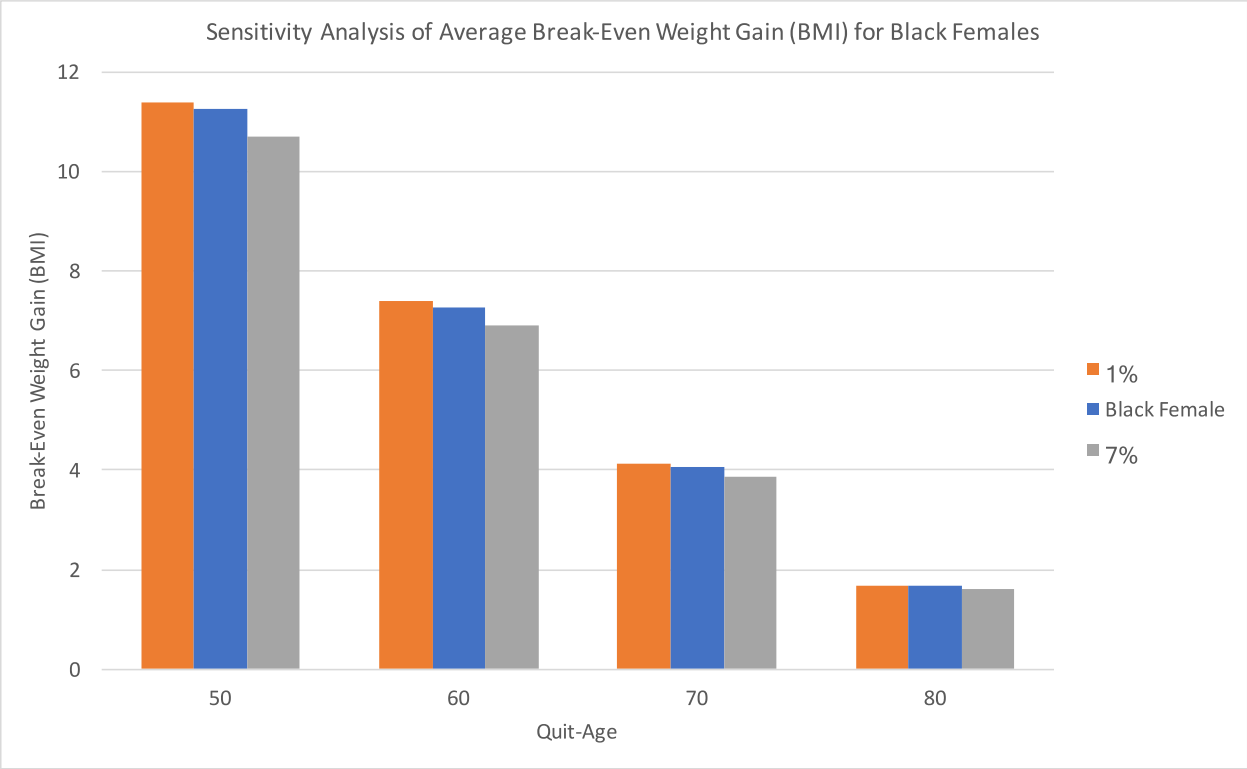
Note. Orange bar = average break-even weight gain in units of BMI for white females with 1% discounting rate; blue bar = 3% discounting rate; grey bar = 7% discounting rate.

Figure 2.E.3 Sensitivity analysis of average break-even weight gain (BMI) for black males under permanent weight gain.



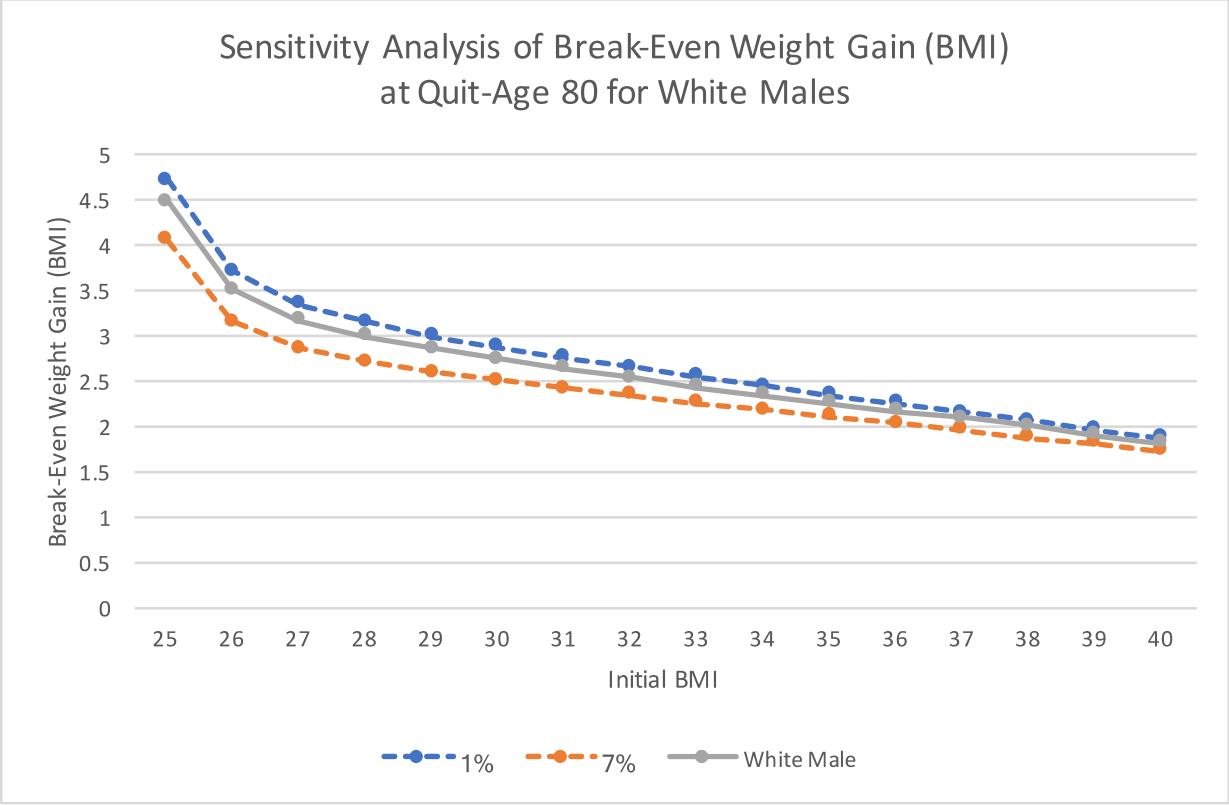
Note. Orange bar = average break-even weight gain in units of BMI for black males with 1% discounting rate; blue bar = 3% discounting rate; grey bar = 7% discounting rate.

Figure 2.E.4 Sensitivity analysis of average break-even weight gain (BMI) for black females under permanent weight gain.



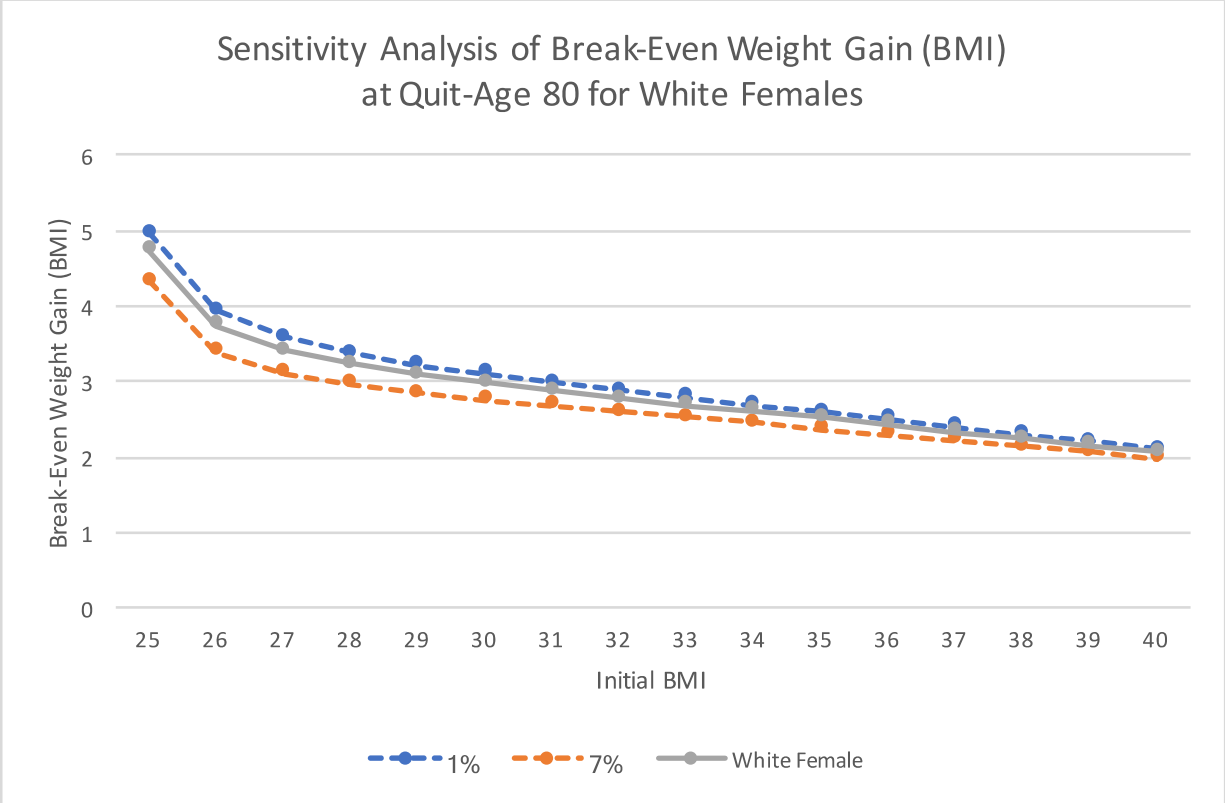
Note. Orange bar = average break-even weight gain in units of BMI for black females with 1% discounting rate; blue bar = 3% discounting rate; grey bar = 7% discounting rate.

Figure 2.E.5 Sensitivity analysis of break-even weight gain (BMI) for white males under diminishing weight gain.



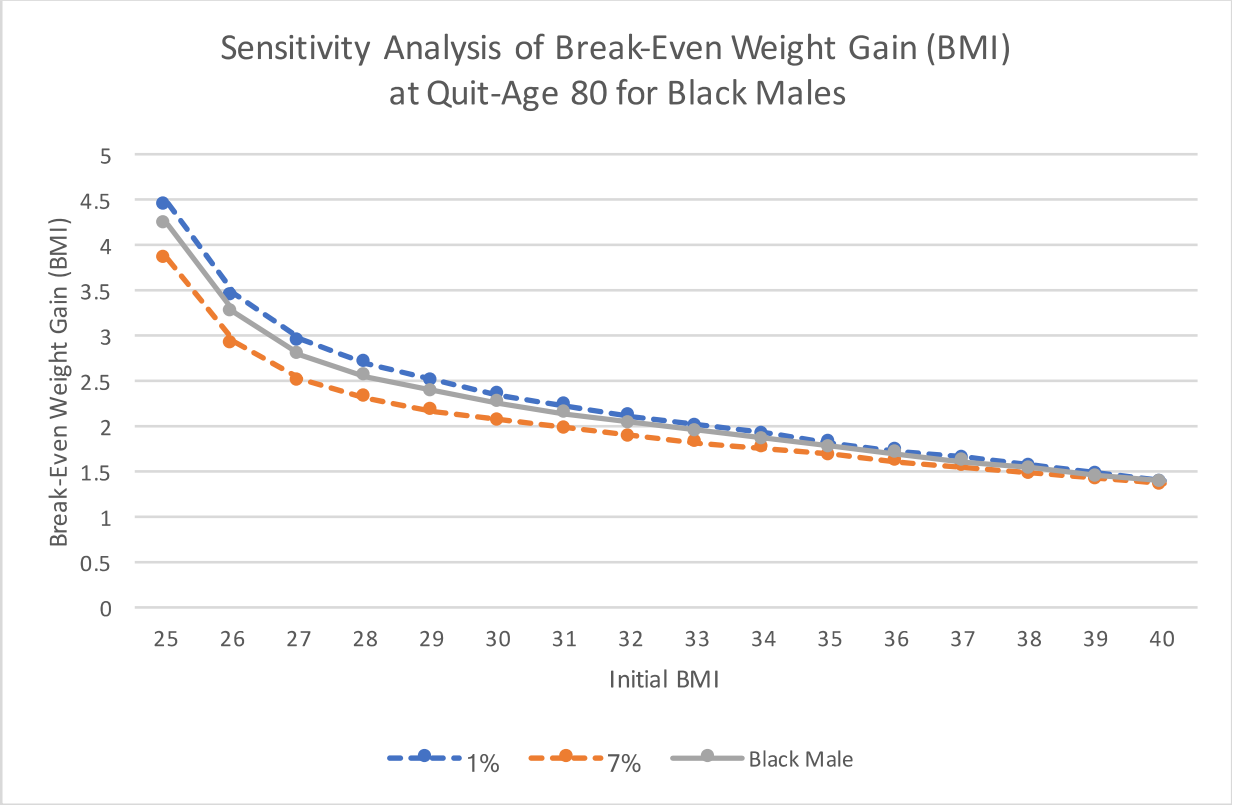
Note. Blue dashed curve = break-even weight gain in units of BMI for white males with 1% discounting rate; grey solid curve = 3% discounting rate; orange dashed curve = 7% discounting rate.

Figure 2.E.6 Sensitivity analysis of break-even weight gain (BMI) for white females under diminishing weight gain.



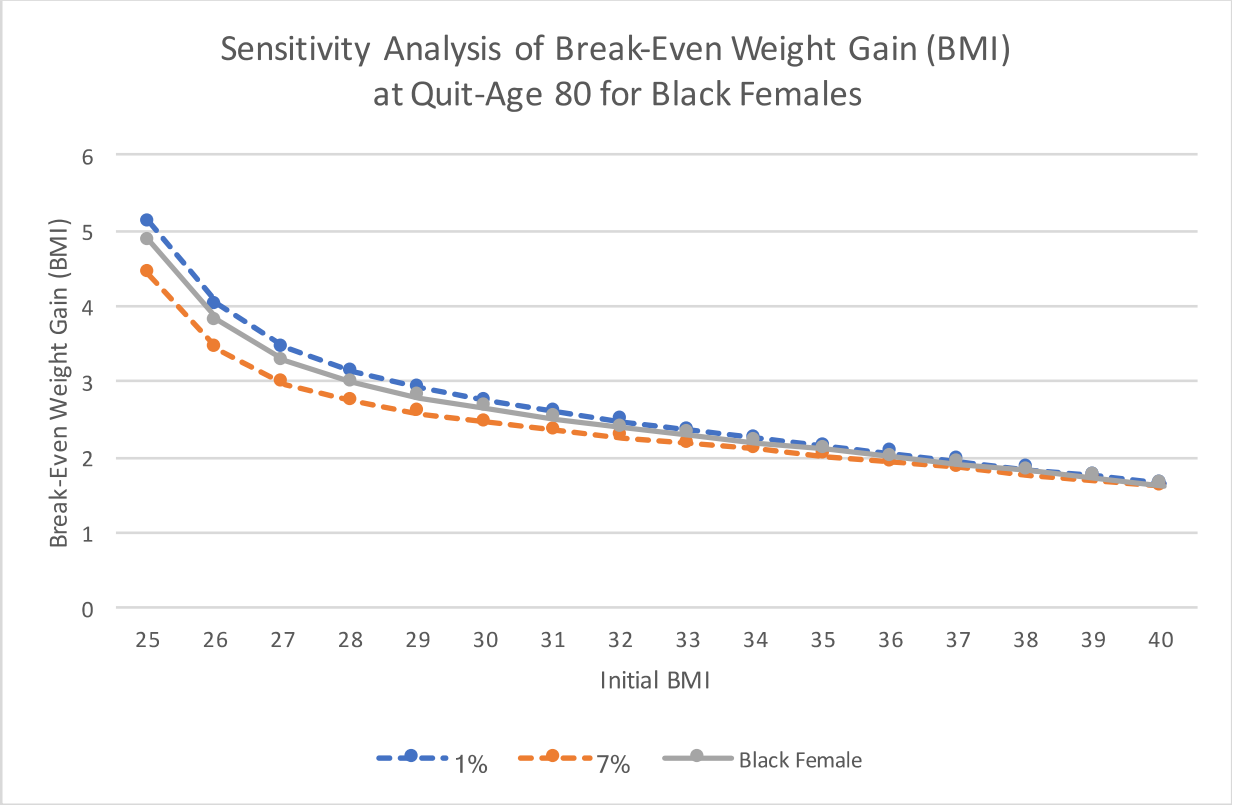
Note. Blue dashed curve = break-even weight gain in units of BMI for white females with 1% discounting rate; grey solid curve = 3% discounting rate; orange dashed curve = 7% discounting rate.

Figure 2.E.7 Sensitivity analysis of break-even weight gain (BMI) for black males under diminishing weight gain.



Note. Blue dashed curve = break-even weight gain in units of BMI for black males with 1% discounting rate; grey solid curve = 3% discounting rate; orange dashed curve = 7% discounting rate.

Figure 2.E.8 Sensitivity analysis of break-even weight gain (BMI) for black females under diminishing weight gain.



Note. Blue dashed curve = break-even weight gain in units of BMI for black females with 1% discounting rate; grey solid curve = 3% discounting rate; orange dashed curve = 7% discounting rate.

Chapter III

Estimating Health Benefits Gained from Reduction in Post-Cessation Weight Gain: An Agent-Based Modelling Approach

3.1 Background

While smoking cessation leads to gains in health benefits, it is often accompanied by a significant increase in body weight as well, referred to as post-cessation weight gain. Concern about post-cessation weight gain has served as a barrier against quitting, especially among women [1-3]. At the same time, post-cessation weight gain attenuates the benefit of smoking cessation. It contributes to an increased risk of type 2 diabetes in the short run, hypertension, and a reduced improvement of lung function [4,5].

Despite the wide variability in post-cessation weight gain, little is known about key predictors of weight gain. There is some evidence that on average women gain more weight than men after quitting [6,7]. Using the Lung Health Study, Ohara et al. found that for quitters with sustained abstinence for 5 years, women gained a mean of 5.2 kg in year 1 and a mean of 3.4 kg in years 1-5 while men gained an average of 4.9 kg in year 1 and a mean of 2.6 kg in years 1-5 [7]. In addition, heavy smokers tend to experience more weight gain [8,9]. Heavy smokers, who smoke more than 25 cigarettes a day, are almost six times more likely to gain more than 13 kg after cessation compared to those who smoke under 15 cigarettes a day [9].

A few studies also examined the impact of baseline Body Mass Index (BMI) on the amount of post-cessation weight gain, but their findings are inconsistent [7,10,11]. Some found that higher baseline BMI is associated with a higher initial post-cessation weight gain [7,11,12], while others discovered a negative or zero association between the two [12-14]. O'Hara reported a higher amount of weight gain after quitting among smokers with higher baseline BMI [7]. Lycett examined POST-CESSATION WEIGHT GAIN in a clinical trial and discovered that obese smokers gained the most weight in 8 years [11]. However, Krukowski and colleagues reported that normal and overweight recent quitters gaining the most significant amounts of weight [10]. Similarly, Veldheer et al. found that normal and overweight smokers gained the most weight after quitting in 10 years [14].

Other factors contributing to higher post-cessation weight gain include lower socio-economic status (SES) and younger quit-age [9,15]. Swan and Carmelli reported that among male former smokers, super-gainers, those who gained more than 13 kg (28.6 lbs) after quitting, were younger and with lower SES [15]. Klesges and colleagues concluded that lower educational attainment is associated with greater initial weight gain [12]. For both genders, people under the age of 55 and who were heavy smokers were associated with higher risk of major weight gain [9].

A literature review conducted by Farley et al. examined various interventions to deal with post-cessation weight gain [16]. These interventions can be divided into three broad categories: 1. Pharmacological treatment; 2. Weight-related behavioral intervention; and 3. Electronic cigarette (EC). Pharmacological treatment can be further categorized into first-line and second-line medications for smoking cessation. First-line pharmacological treatments include nicotine

replacement therapies (NRTs), bupropion, and varenicline. Some second-line treatments are clonidine and nortriptyline. These treatments block the positive reinforcing effect of nicotine and prevent or reduce nicotine withdrawal symptoms [17]. However, these medications appear to delay, rather than prevent post-cessation weight gain. A significant reduction in weight gain is discovered at the end of the treatment but there is no evidence that these treatments reduced weight in the long run at 6 or 12 months [18]. NRT, bupropion, and varenicline were reported to reduce weight gain by 0.5 kg, 1.1 kg and 0.4 kg, respectively, at the end of the treatment [16].

Weight-related behavioral interventions include personalized weight management support, exercise interventions and energy restriction or healthy eating advice [16,18,19]. Most behavioral interventions aim to minimize post-cessation weight gain by limiting calorie intake [20,21], enhancing energy expenditure [22,23] or a combination of both [24-26]. A systematic review done on behavior interventions by Spring et al. found significant effect in the short run [27]. Farley et al. reported that both personalized weight management support and exercise interventions reduced weight gain significantly at 12 months, but the short run reduction was not significant [16].

Another innovative approach to deal with post-cessation weight gain is to use alternative tobacco product such as EC. EC is a battery-operated device designed to vaporize an e-liquid by heating an element that generates an inhalable aerosol. This process is referred to as vaping. EC does not contain tobacco, do not create smoke and do not rely on combustion to operate [28]. Many smokers use EC to help quit or reduce smoking [29,30]. Recent studies show that EC attenuates tobacco withdrawal symptoms as effectively as NRT [31,32l]. Russo et al. investigated changes in body weight in smokers who quit or reduced their smoking cessation substantially by switching to EC

use [28,33]. They found that quitters gained a significant 4.8% weight from baseline at 12 months after cessation while weight gain for EC users was only 1.5% of baseline [33]. ECs is considered as a tobacco harm reduction mechanism. Smokers who switch to EC achieve large but not full health gains of cessation [34]. In addition, they are more likely to quit [35]. Among dual users, 46% quit smoking altogether after 1 year [29,31].

In this study, I estimate health benefits gained from reduction in post-cessation weight gain where three different interventions are implemented. My main outcome measures are life-years saved, cumulative survival probability and obesity prevalence at the population level. These interventions are pharmacological treatment, EC and behavioral intervention. I first simulate a nationally representative sample of adult smokers in the US via the National Health Interview Survey (NHIS) in 2017. Then I apply interventions dealing with both smoking cessation rates and post-cessation weight gain, assuming a 100% participation rate. The model mentioned above is implemented via an agent-based model to account for population heterogeneity, where differential mortality is applied based on demographic characteristics and weight trajectories. The model tracks BMI and survival information for each simulated smoker over 20 years as outcome measures. The agent-based model is implemented via Python programming.

3.2 Methods

Study Population

I obtained demographic information of adult smokers in the US from the NHIS 2017 dataset. The NHIS has monitored the health of the US population since 1957. NHIS data are collected on a broad range of health topics by the U.S. Census Bureau. A nationally representative sample of

households are surveyed each year. This dataset is open to public and I mapped the 2017 population onto the agent-based model at baseline.

I started with 4,015 smokers, excluded individuals who have missing BMI values (121); missing smoking intensity (81); under 18 years-old (15); missing race information (203); missing height values (238); and missing data on educational achievement (16). My final sample includes 3341 adult smokers. In addition to adopting the NHIS sample as the initial population, I also used bootstrap method to generate random samples with 10,000 smokers where survey weights from the NHIS were incorporated. I refer to these two samples as the NHIS sample and the random sample. Table 3.1 provides a summary of baseline characteristics from both the NHIS sample and the random sample. In both samples, average age initially is in the mid to late 40s. There are slightly more males among smokers, 55.15% (random sample) and 51.63% (NHIS sample). The majority of the smokers are white, 85.5% vs 86.95%. Both samples have around 30% of the population that are obese. And majority of the smokers have finished high school (84.27% vs 83.12%). One parameter I added in the sample is POST-CESSATION WEIGHT GAIN. I assume it has a normal distribution with a mean of 10.3 lbs per the results from Aubin et al. [36].

Table 3.1 Baseline characteristics of adult smokers in the simulated cohort vs the National Health Interview Survey, 2017.

Lifetime weight trajectory

The lifetime weight trajectories for current smokers are calculated, adjusting for age, gender, race, BMI from previous survey, and SES (high school graduate or not). I used 12 waves of the Health and Retirement Study (HRS) from 1992 to 2014. HRS is a longitudinal panel study that surveys a

representative sample of the US population every two years. It is conducted by the Survey Research Center at the University of Michigan and sponsored by the National Institute on Aging. At the baseline in 1992, a total of 12,652 individuals from 7,702 households participated. My sample includes only this initial HRS cohort who joined the study in 1992. A few exclusion criteria are applied to obtain my final sample. The flowchart below in Figure 3.1 illustrates the process. I tracked all twelve waves of data (1992 till 2014) to investigate how BMI changes with respect to smoking behavior and demographic variables over time.

Figure 3.1 Flowchart for selecting eligible participants in the final sample from the Health and Retirement Study.

My final sample includes 9,606 unique individuals and 69,745 person-year observations. At baseline, the average age is 55.22 years, 43.04% of participants are male, 80.23% of them are white and the average BMI is 26.99. The obesity rate is 22.93%, 81% are married and around 70% have degrees of high school or above. See Appendix 3.A for more details.

To estimate the lifetime weight trajectory of individuals adjusting for smoking behaviors and demographic information, I ran linear regressions to predict the percentage change of BMI between any two consecutive surveys adjusting for individual level clustering. I used Stata version 14.0 (StataCorp LP, College Station, TX) to conduct this analysis. My dependent variable is the percentage change of BMI between any two consecutive surveys and my independent variables include age, gender, race, SES, and BMI from previous survey. Regression results can be found in Appendix 3.B.

Mortality Rate

An article published by the Global BMI Mortality Collaboration assessed the impact of BMI on all-cause mortality [21]. Using their hazard ratio estimates for various BMI levels controlling for gender, I calculated the RR for obesity as a function of BMI for both genders. For BMI over 25, mortality rate increases log-linearly, adjusting for gender [21]. To calculate the RR of mortality adjusting for smoking behaviors, I employed the all-cause mortality RR calculated in Mendez and Warner's work [22]. They derived the RR for current and former smokers as a function of age, gender, and years quit via logistic regressions.

Another important piece is the all-cause mortality rate for the control group. In Mendez and Warner's work, the baseline risk used is the mortality rate for never-smokers [22]. For obesity-related RR, the hazard ratio is 1 for normal-weight individuals with a BMI between 18.5 and 25. The baseline mortality rate in this study is the one for normal-weight never-smokers. From a recent publication of the National Vital Statistics Reports, I obtained annual mortality rates in the US by race and gender in 2016 [23]. I then combined the mortality rates with a group prevalence weight calculated from the National Health Interview Survey (NHIS) to derive estimates for normal-weight never-smokers. I counted the impact of smoking and obesity on all-cause mortality as additive per the results from Mehta and Preston's work [24]. The age-specific annual mortality rates by gender and race from age 40 to 100 for normal-weight never-smokers can be found in Table 3.2.

Table 3.2 Age-specific annual mortality rates for normal-weight never-smokers by gender and race.

3.3 Model

The tool I employed to investigate the effectiveness of these interventions is agent-based modeling (ABM). ABM, a relatively new method in the social science arsenal, has enabled scholars to investigate behaviors and social interactions at individual or organizational levels. ABM can incorporate various individual and environmental characteristics into the model to account for population heterogeneity. In this study, ABM accounts for population heterogeneity by incorporating demographic information for each simulated agent. Through ABM, I first simulated a nationally representative sample of the US adult smokers using NHIS data in 2017. Then I evaluated the impact of three interventions on this simulated population. My outcome measures are life-years saved and obesity prevalence over 20 years at the population level.

Initially in my model at Year 1, all individuals are smokers. Here one time-step in the model is one year. Year 1 (baseline) is 2017 and Year 2 is 2018. At each time-step in the model, I simulated potential smoking behavioral changes. If smokers continue smoking, their weight trajectories follow the ones I predict via regression models. If smokers quit, they experience a post-cessation weight increase. This weight increase remains permanent on top of their weight trajectories assuming if they were continuing smokers. Based on the literature review conducted by Aubin et al., the average post-cessation weight gain in the population is around 10.3 lbs with a variance of 0.79 lbs.

I tracked all simulated individuals over a 20-year horizon, aka 20 time-steps. I examined the survival rate by counting the number of individuals who survive after each time step. Age, sex and race adjusted all-cause mortality rate, together with relative risk of BMI and smoking status, are applied to model the death event for each simulated agent at each time-step. I ran a total of 200

rounds of simulation for each data point to account for randomness in the model. The results presented are averages across all 200 rounds of simulation. A step-by-step description of the model can be found in the Appendix 3.C.

Interventions

In order to investigate the effectiveness of interventions, I created a control group with no interventions and implement a total of three interventions on the same initial population. These interventions adopted are pharmacotherapy (bupropion treatment), behavioral intervention (physical activity program) and EC.

Control Group: smoking cessation only
Intervention I: smoking cessation + pharmacotherapy (bupropion)
Intervention II: smoking cessation + behavioral intervention (physical activity program)
Intervention III: smoking cessation + electronic cigarette

A brief summary of these interventions can be found in Table 3.3 below. I selected these interventions because they are randomized control trials (RCTs) that provide more accurate estimates of intervention effectiveness. A more detailed description of these RCTs can be found in Appendix 3.D. The pharmacotherapy program implements bupropion as a pharmacotherapy to reduce post-cessation weight gain. The effectiveness estimation is from a 7-week RCT conducted by Nides et al. [37]. The behavioral intervention is a physical activity program where individuals enroll for 9 weeks with structured and lifestyle components [38]. Intervention EC helps smokers to switch to electronic cigarette. The treatment group includes both dual users (cigarettes + electronic cigarettes) and exclusive users (electronic cigarette only). There is no significant difference between these two groups considering weight gain at week 52 [33].

Table 3.3. Summary of interventions.

I converted the post-cessation weight gain values in these interventions to relative weight gains in percentage points, using quitters in the control group as the reference group. Similarly, I derived the impact of interventions on smoking cessation rate to relative values in percentage points [33,37,38]. Table 3.4 below summarizes the parameter values for all three interventions. For example, 1.29 for pharmacotherapy under smoking cessation rate means pharmacotherapy intervention increases the smoking cessation rate by 29 percentage points, compared with the control group. 0.47 for electronic cigarette under post-cessation weight gain implies that smokers who switch to electronic cigarette experience 53 percentage points reduction in post-cessation weight gain at the end of one year. Since we know the population average smoking cessation rate is around 4.5% [39] and the weight gain has a normal distribution with mean of 10.3 lbs [36], I adjusted these rates according to the simulated interventions in the model. In Table 3, pharmacotherapy only increases smoking cessation rate but does not affect post-cessation weight gain in the long run. Physical activity, on the other hand, helps with post-cessation weight increase but does nothing to smoking cessation rate. Electronic cigarette seems to be the most desirable intervention since it raises smoking cessation rate and reduces weight gain post-cessation.

Table 3.4 Value of parameters in the model estimating effectiveness of interventions.

Scenarios

I modeled two different scenarios: Scenario I vs Scenario II. In Scenario I, all smokers quit smoking in Year 1 and do not relapse. In this scenario, the model only assesses the impact of interventions via their reductions in post-cessation weight gain. Since the cessation rate is 100% across interventions, Scenario I does not consider the impact of these interventions on smoking

cessation. In Scenario II, a population level quit rate of 4.5% is added to the model [39]. In Scenario II, interventions not only decrease weight increase but also increases smoking cessation rate, generating a more complex impact on the population. These interventions are implemented in Year 1 only, where they affect both the smoking cessation rate and post-cessation weight increase.

3.4 Results

In Scenario I where I assumed the smoking cessation rate to be 100% in Year 1, since pharmacotherapy has no effect on post-cessation weight gain, status quo with no interventions generate the same results as pharmacotherapy. I thus omit the results from pharmacotherapy and only list status quo with two other interventions (physical activity and EC). Table 3.5 presents the cumulative survival probability over twenty years under the random sample and the NHIS sample. Under the random sample, both EC and physical activity interventions increased cumulative survival probability and the increases are statistically significant. In 5 years, if smokers switch to electronic cigarette, their survival rate will increase by around 0.23 percentage point. If they participate in physical activity interventions, their survival rate will increase by 0.04 percentage point. By the end of twenty years, differences grow to 1.19 percentage points for EC and 0.16 percentage point for physical activity intervention. Similar results can be found with the NHIS sample. EC intervention on average generates higher cumulative survival probability compared to physical activity or the status quo. One minor difference is that in the NHIS sample, the survival probability under physical activity is not statistically different from the status quo at Year 5, but it is post Year 10. Although these significant changes are not large, considering we have around 34 million adult smokers in the US currently, the potential life-years saved is huge.

Table 3.5. Cumulative survival probability in Scenario I, where all smokers quit.

Table 3.6 shows the variation of average obesity prevalence over 20 years. In Table 6 with the random sample, EC and physical activity both reduce obesity prevalence in the population significantly since Year 5. At Year 5, obesity prevalence is reduced by 6.68 percentage points via electronic cigarette intervention and by 4.22 percentage points via physical activity. At the end of twenty years, these reductions are further increased to 9.68 and 5.26 percentage points respectively.

Table 3.6. Average obesity prevalence in Scenario I, where all smokers quit.

Figure 3.2 illustrates the cumulative survival probability by year comparing status quo with two interventions with the random sample. EC intervention generates a bigger impact than physical activity. The impact of these interventions on average obesity prevalence with 100% cessation rate is shown in Figure 3.3. The initial jump from Year 1 to Year 2 is the result of cessation. It is obvious that both interventions decrease obesity prevalence significantly. Results regarding the NHIS sample can be found in Appendix 3.E.

Figure 3.2. Average cumulative survival probability in one-year increments under the random sample in Scenario I, where all smokers quit.

Figure 3.3 Average obesity prevalence in one-year increments under the random sample in Scenario I, where all smokers quit.

Then I examined the outcomes from Scenario II in Table 3.7, where the cessation rate is 4.5% and the impact of interventions on smoking cessation are added. In both random sample and the NHIS sample, pharmacotherapy generates minor improvement to the cumulative survival probability. This improvement is not statistically significant in the short run. Similarly, physical activity creates insignificant minor improvement until Year 20, where the survival probability is increased by 0.11

and 0.13 percentage point in the random and NHIS sample, respectively. EC seems to be the dominant intervention. It leads to higher cumulative survival probability and the improvement is significant post Year 10. By the end of twenty years, EC intervention increases the survival probability by approximately 1.2 percentage points. Translating these results into life-years saved in Table 3.8, we find that EC intervention could save 3,500 life-years in 5 years using the random sample. The estimated life-years saved increases to 238,000 in 20 years, which is a significant amount.

Table 3.7. Cumulative survival probability in Scenario II, where status quo cessation rate is 4.5%.

Table 3.8. Life-Years Saved in Scenario II compared with status quo, where status quo cessation rate is 4.5%.

Table 3.9 reveals the changes of average obesity prevalence over twenty years under different interventions. Both EC and physical activity are statistically significant at reducing obesity prevalence while pharmacotherapy actually increases the obesity prevalence. This is not unexpected. Pharmacotherapy is effective at increasing smoking cessation rate but does not help with post-cessation weight increases. With a growing number of quitters who might become obese via post-cessation weight gain, it is not surprising that pharmacotherapy intervention increases obesity prevalence and cumulative survival probability simultaneously. The differences in one-year increments are illustrated via figures in Appendix 3.F.

Table 3.9 Average obesity prevalence in Scenario II, where status quo cessation rate is 4.5%.

3.5 Discussion

The health message given to smokers currently is that it is never too late to quit, without addressing the issue of post-cessation weight gain. The agent-based model established in this paper attempts to estimate the potential health benefits that could be gained if post-cessation weight gain is tackled via different interventions. In general, my results imply that all of these interventions improve cumulative survival probability significantly in the long run, with EC being the most effective.

Another interesting finding is that smoking cessation can bring improvement to survival probability but can also increase the obesity prevalence. In Scenario II, when smokers participate in the pharmacotherapy intervention, they have a higher change to quit but no reduction in post-cessation weight gain. The result shows a higher survival probability and a higher obesity prevalence. However, if smokers enroll in interventions that address post-cessation weight increase, they experience the better of both, higher survival probability and lower obesity prevalence. The findings show the perils of addressing these two major public health problems independently. The interaction between smoking behavior and body weight change makes it important for us to look at both obesity and smoking as one big problem.

Findings from the model suggest that EC is very promising in increasing survival rate and reducing obesity prevalence. Quitting smoking and switching to electronic cigarette is considered as a harm reduction mechanism. Harm reduction in public health refers to the framework where health policies focus on decreasing the harmful consequences of drugs without fully eliminating the use [40]. ECs appear to be far less harmful than cigarettes, but the long-term effect remains unknown. For example, the 2018 National Academies of Sciences, Engineering, and Medicine Report found that EC use is positively associated with an increase of smoking initiation among adolescents [41].

At the same time, the whole harm reduction strategy to tobacco control stays a controversial topic. While some are fond of the idea of harm reduction and view it as a promising progress towards elimination, others have concerns over lack of regulation, manipulation from tobacco companies and potential health disparities in the future. While this study recognizes electronic cigarette as a promising intervention to deal with POST-CESSATION WEIGHT GAIN for adult smokers, more research is needed to investigate long-term effects of electronic cigarette and update model results accordingly.

Although physical activity intervention showed only marginal benefits in the model, it is potentially more beneficial due to its positive externalities. For example, physical activity helps to build muscle mass, promotes cardiometabolic wellness and improves cognitive performance [42,43]. In this model, I only captured impact of interventions via smoking cessation or post-cessation weight change without including other potential mechanisms such as life style or diet change.

This study also has some limitations. First, I assumed a normal distribution of weight increase and assigned it randomly to simulated individuals. Previous studies have shown that post-cessation weight increase is correlated with factors such as smoking intensity and gender. In the future, I plan to incorporate these correlations in my model to generate more accurate estimates of post-cessation weight gain for individuals. Another limitation is the assumption of 100% participation rate in interventions. In the future, I plan to relax this assumption to allow more flexibility among the population. Better estimation of participation rates in the population can help us derive more accurate population-level results. The post-cessation weight gain mechanism is another limitation

of this study. Currently, the community has not yet reached an agreement on whether weight gain post-cessation diminishes or not [44,45]. In this model I assumed a permanent weight gain from cessation. Future research is needed to look into how smokers gain weight post-cessation.

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Tables and Figures

Table 3.1 *Baseline characteristics of adult smokers in the simulated cohort vs the National Health Interview Survey, 2017.*

Characteristics	**N = 10,000** (Mean ± sd)	**N = 3,341** (Mean ± sd)
Age (years)	46.06 ± 15.38	48.4 ± 15.59
Male (%)	55.13	51.63
White Race (%)	85.5	86.95
Body Mass Index (BMI)	27.81 ± 6.67	27.38 ± 5.5
Normal Weight (BMI < 25 kg/m^2)	36.7%	37.44%
Overweight (25 kg/m^2 ≤ BMI < 30 kg/m^2)	34.71%	34.51%
Obese (BMI ≥ 30 kg/m^2)	27.47%	28.05%
Height (inch)	67.32 ± 3.94	67.26 ± 3.91
Post-Cessation Weight Gain (lbs)	**10.3 ± 0.79**	**10.3 ± 0.79**
Intensity of Smoking (%)		
Light Smoker (1 – 10 cig/day)	61.01	58.72
Moderate Smoker (11 – 20 cig/day)	11.75	12.15
Heavy Smoker (≥20 cig/day)	27.24	29.12
Education (%)		
Less than High School	15.73	16.88
High School Graduate and Above	84.27	83.12

Table 3.2 Age-specific annual mortality rates for normal-weight never-smokers by gender and race.

Age	White Male (%)	White Female (%)	Black Male (%)	Black Female (%)
40-44	0.12	0.10	0.18	0.14
45-49	0.16	0.14	0.22	0.19
50-54	0.25	0.20	0.36	0.30
55-59	0.36	0.30	0.53	0.43
60-64	0.53	0.43	0.86	0.62
65-69	0.73	0.63	1.12	0.86
70-74	1.13	1.05	1.57	1.28
75-79	1.86	1.79	2.20	1.99
80-84	3.22	3.19	4.08	3.29
85-99	7.64	8.87	8.23	7.47

Note. We assume the maximum life span is 100 years.

Table 3.3 Summary of interventions.

Intervention	Year	Sample Size	Control Group	Treatment Group	Weight Gain for quitters in a year
Pharmacotherapy (Bupropion)	2000-2003	353 (with clinic visits)	Placebo for 7 weeks.	Bupropion hydrochloride was dosed for 7 weeks, with titration from 150 mg once daily (days 1-3) to 150 mg twice daily through week 7.	No difference in 52 weeks.
Behavioral Intervention (Physical Activity Program)	2002-2006	481	9 weeks of smoking cessation	9 weeks of smoking cessation + 9 weeks of physical activity program.	Control: 6.2 kg Treatment: 4.4 kg
Electronic Cigarette	2012-2015	223	Quitters who reported sustained smoking abstinence after completing a cessation program	Electronic Cigarette users (including dual users).	Control: 4.8% from baseline Treatment: 1.5% from baseline

Table 3.4 Value of parameters in the model estimating effectiveness of interventions.

	Smoking Cessation Rate	Post-Cessation Weight Gain
Pharmacotherapy	1.29	1
Electronic Cigarette	2.38	0.47
Physical Activity	1	0.71

Note. Effectiveness is estimated in terms of ratio where the control group is status quo with no interventions.

Table 3.5 Cumulative survival probability in Scenario I, where all smokers quit.

Cumulative Survival Probability	**Random Sample**			**NHIS Sample**		
	Status Quo	E-cig	Physical Activity	Status Quo	E-cig	Physical Activity
5 years (%)	94.94 (0.0036)	95.24*** (0.0035)	94.97 (0.0037)	95.6 (0.0018)	95.83*** (0.002)	95.64** (0.0019)
10 years (%)	87.67 (0.005)	88.4*** (0.0047)	87.77** (0.0055)	88.24 (0.0027)	89.79*** (0.0028)	89.31*** (0.0028)
15 years (%)	79.16 (0.0063	80.22*** (0.0052)	79.38*** (0.0065)	81.68 (0.0035)	82.56*** (0.0032)	81.8*** (0.0034)
20 years (%)	69.53 (0.0066)	70.78*** (0.0057)	69.79*** (0.0069)	72.93 (0.0039)	74.12*** (0.0037)	73.09*** (0.0042)

Standard errors are in parentheses
** p < 0.10, ** p<0.05, and *** p<0.01.*

Table 3.6 Average obesity prevalence in Scenario I, where all smokers quit.

Obesity Prevalence	**Random Sample**			**NHIS Sample**		
	Status Quo	E-cig	Physical Activity	Status Quo	E-cig	Physical Activity
5 years (%)	44.34 (0.00096)	37.66*** (0.00097)	40.12*** (0.00091)	41.87 (0.0019)	35.94*** (0.0018)	38.43 (0.0019)
10 years (%)	52.6 (0.0016)	43.76*** (0.0015)	48.25*** (0.0016)	50.19 (0.0029)	41.7*** (0.0027)	45.62** (0.0031)
15 years (%)	58.91 (0.0021)	49.79*** (0.002)	53.98*** (0.0022)	55.58 (0.004)	46.66*** (0.0037)	50.61*** (0.0039)
20 years (%)	62.99 (0.0027)	53.31*** (0.0024)	57.73*** (0.0031)	59.1 (0.0051)	49.44*** (0.0045)	53.66*** (0.0051)

Standard errors are in parentheses
** p < 0.10, ** p<0.05, and *** p<0.01.*

Table 3.7 Cumulative survival probability in Scenario II, where status quo cessation rate is 4.5%.

Cumulative Survival Probability	**Random Sample**				**NHIS Sample**			
	Status Quo	Pharmaco-therapy	E-cig	Physical Activity	Status Quo	Pharmaco-therapy	E-cig	Physical Activity
5 years (%)	95.69 (0.002)	95.69 (0.0019)	95.7 (0.0019)	95.68 (0.002)	95.11 (0.0035)	95.1 (0.0034)	95.14 (0.0036)	95.12 (0.0036)
10 years (%)	89.47 (0.0031)	89.64** (0.0026)	89.8*** (0.0027)	89.58 (0.0031)	88.2 (0.0048)	88.23 (0.0052)	88.45*** (0.005)	88.19 (0.0056)
15 years (%)	82.58 (0.0035)	82.7** (0.0034)	83.25*** (0.0034)	82.66 (0.0034)	80.19 (0.0062)	80.28 (0.006)	80.88*** (0.006)	80.27 (0.0063)
20 years (%)	74.64 (0.0039)	74.89*** (0.0037)	75.89*** (0.0038)	74.75*** (0.0036)	71.24 (0.0066)	71.49*** (0.006)	72.5*** (0.0063)	71.37** (0.0071)

Standard errors are in parentheses

** p < 0.10, ** p<0.05, and *** p<0.01.*

Table 3.8. Life-Years Saved in Scenario II compared with status quo, where status quo cessation rate is 4.5%.

Life-Years Saved	**Random Sample** (N = 10,000) Pharmacotherapy	Electronic Cigarette	Physical Activity
5-year	1500 (95)	3500*** (85)	1500 (100)
10-year	21000** (260)	41000*** (270)	20000 (300)
15-year	19500** (465)	103500*** (480)	9000 (480)
20-year	46000*** (680)	238000*** (700)	18000* (760)

Standard errors are in parentheses
** p < 0.10, ** p<0.05, and *** p<0.01.*

Table 3.9 Average obesity prevalence in Scenario II, where status quo cessation rate is 4.5%.

Average Obesity Prevalence	Random Sample				NHIS Sample			
	Status Quo	Pharmaco-therapy	E-cig	Physical Activity	Status Quo	Pharmaco-therapy	E-cig	Physical Activity
5 years (%)	33.25 (0.0016)	33.76*** (0.0017)	33.35*** (0.0015)	32.65*** (0.0014)	32.55 (0.0028)	33.1*** (0.0029)	32.5** (0.0022)	31.92*** (0.0022)
10 years (%)	40.68 (0.0024)	41.87*** (0.0025)	39.81*** (0.002)	39.33*** (0.0022)	39.36 (0.0045)	40.63*** (0.0045)	38.63*** (0.0032)	37.86*** (0.0041)
15 years (%)	48.52 (0.003)	50.21*** (0.0029)	46.66*** (0.0022)	45.63*** (0.0025)	40.63*** (0.0045)	48.18*** (0.0055)	44.87*** (0.004)	44.06*** (0.0045)
20 years (%)	54.32 (0.0037)	56.01*** (0.0034)	51.21*** (0.0026)	51.23*** (0.0031)	51.32 (0.0055)	52.92*** (0.006)	48.21*** (0.0047)	48.15*** (0.0054)

Standard errors are in parentheses
** p < 0.10, ** p<0.05, and *** p<0.01.*

Figure 3.1 Flowchart for selecting eligible participants in the final sample from the Health and Retirement Study.

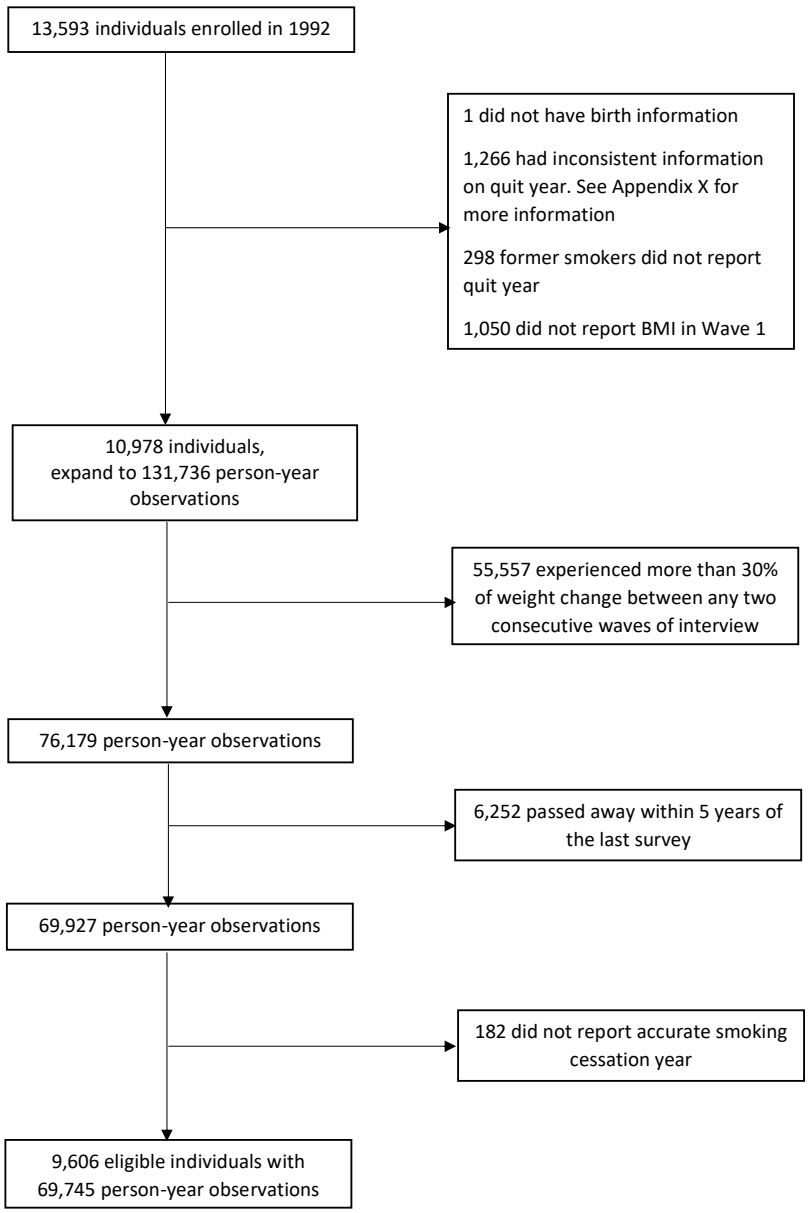
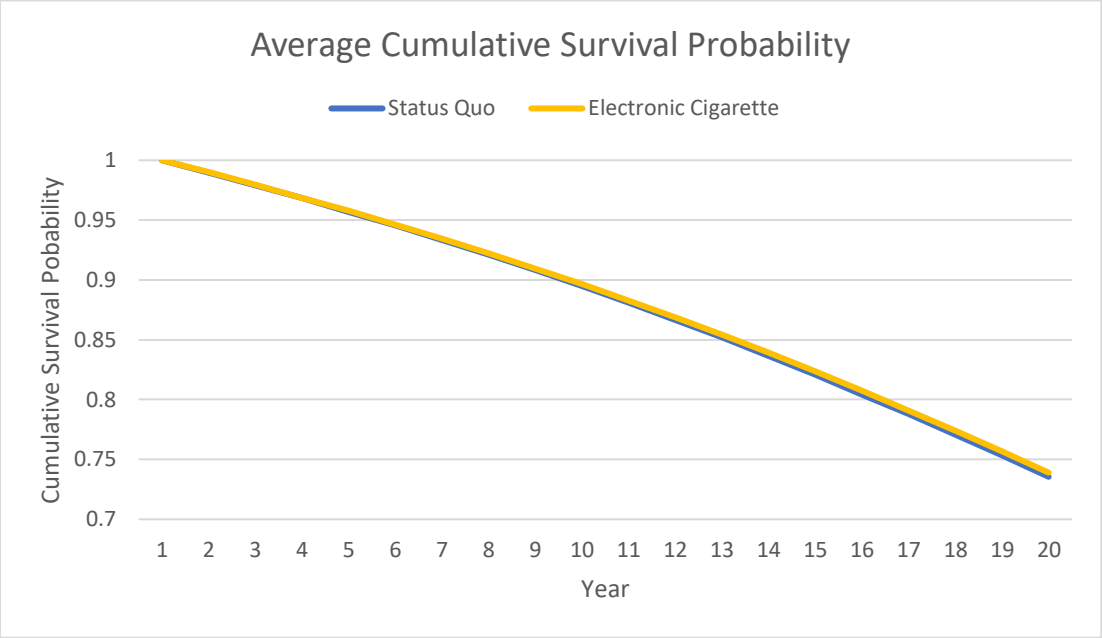


Figure 3.2. Average cumulative survival probability in one-year increments under the random sample in Scenario I, where all smokers quit.



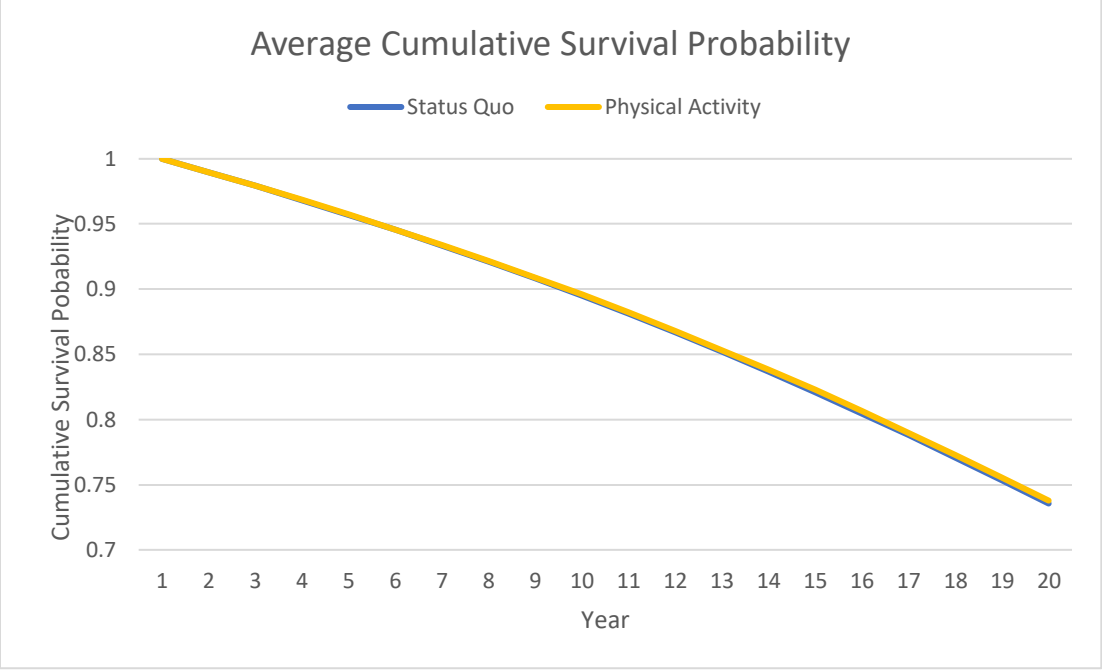
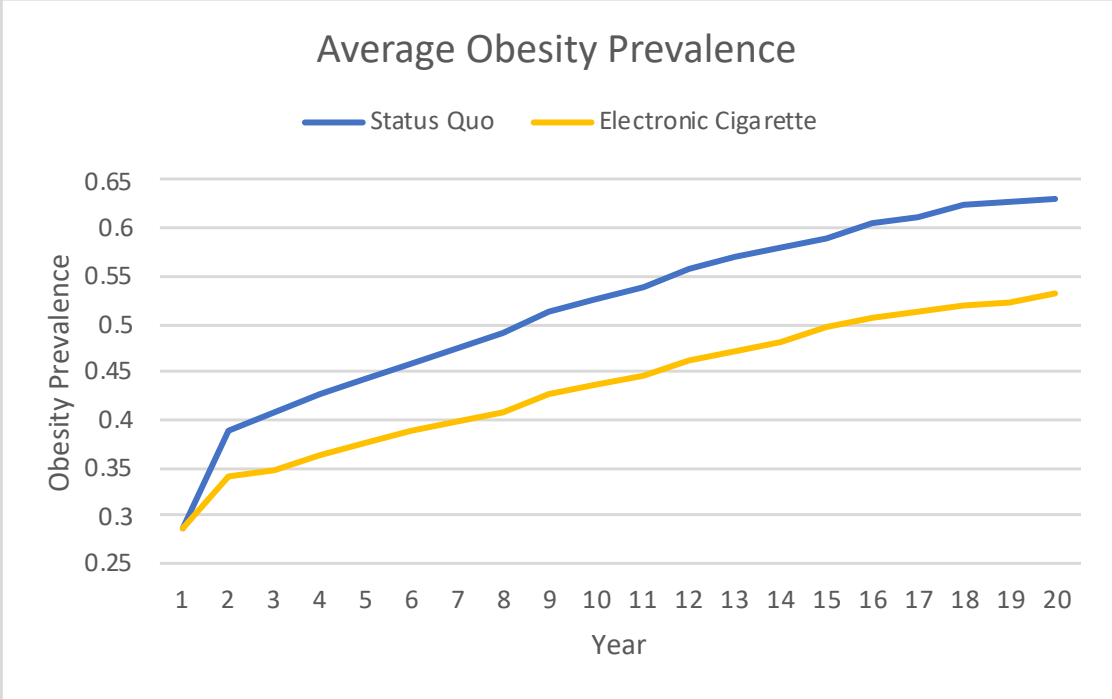
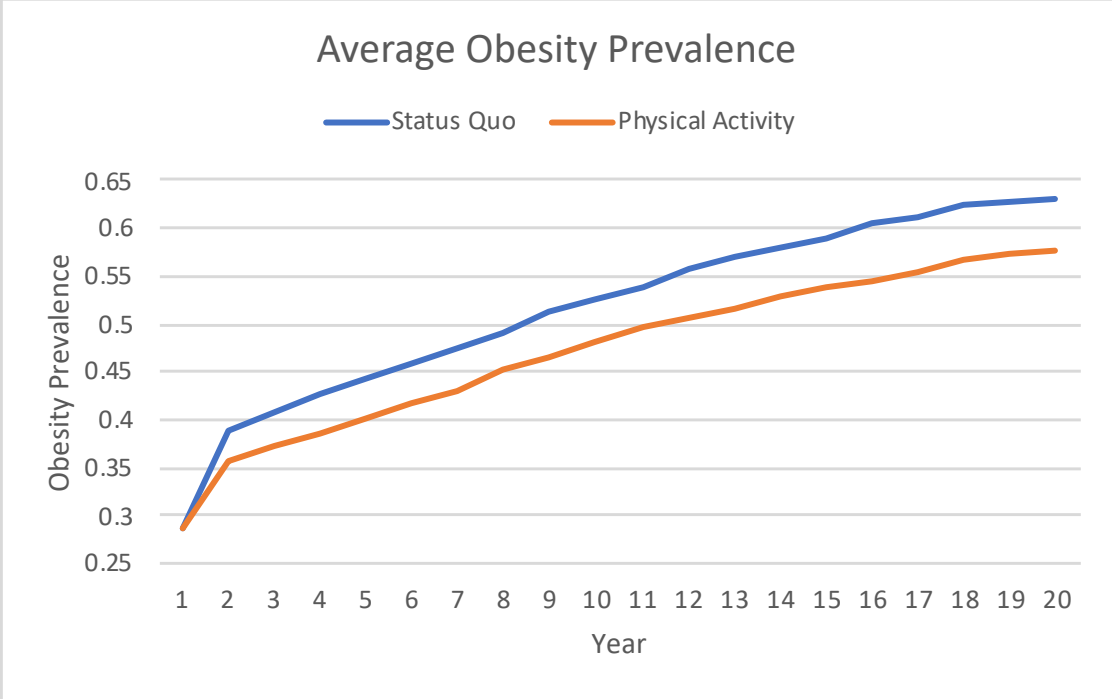


Figure 3.3 Average obesity prevalence in one-year increments under the random sample in Scenario I, where all smokers quit.





Appendices

Appendix 3.A Baseline characteristics.

Table 3.A.1 Baseline characteristics of the sample population, Health and Retirement Study 1992.

Characteristics	N = 8,958
Age (years)	55.22 ± 5.55
Male (%)	43.03
White Race (%)	80.23
Weight (kg)	77.62 ± 16.62
Body Mass Index (BMI)	26.99 ± 5.03
Normal Weight (BMI < 25 kg/m^2)	36.68
Overweight (25 kg/m^2 ≤ BMI < 30 kg/m^2)	40.39
Obese (BMI ≥ 30 kg/m^2)	22.93
Height (m)	1.69 ± 0.099
Smoking status (%)	
Current Smoker	30.53
Former Smoker	25.11
Never Smoker	44.36
Marital Status (%)	
Married/Partnered	81.16
Single	2.88
Divorced/Widowed	15.95
Education (%)	
Less than High School	30.22
High School Graduate	33.05
Some College	19.41
College and Above	17.31

Appendix 3.B Regression.

Table 3.B.1 Regression results of change in BMI (%) for never smokers and current smokers.

Change in BMI (%) between any two consecutive surveys	Never Smokers (N = 33,458)	Current Smokers (N = 13,607)
Age	-0.00063^{***}	-0.00057^{***}
	(0.00004)	(0.0000766)
Sex	0.00132^{**}	0.0007
	(0.00059)	(0.0010777)
SES	0.00091	0.00007
	(0.00075)	(0.00118)
Race	-0.00108^{*}	-0.00239^{**}
	(0.0006)	(0.00107)
BMI (previous survey)	-0.00167^{***}	-0.00188^{***}
	(0.00008)	(0.00014)

Standard errors are in parentheses.
* p < 0.10, ** p<0.05, and *** p<0.01.

Appendix 3.C Intervention Descriptions

1. Pharmacotherapy Intervention [37]

This randomized, double-blind, parallel-group, placebo- and active-controlled clinical trial was conducted at 7 US sites from February 2000 to January 2003. Randomized subjects received 1 of 3 varenicline tartrate dose regiments, sustained-release bupropion hydrochloride or matched placebo. Bupropion hydrochloride was dosed for 7 weeks, with titration from 150 mg once daily (days 1-3) to 150 mg twice daily through week 7. Subjects who volunteered to join the non-drug treatment received clinic visits at week 12, 24 and 52. A total of 638 individuals participated in the randomized clinical trial and 353 completed all clinic visits, where 66 were in the placebo group and 68 were in the bupropion treatment group.

Mean weight gain from baseline to week 7 was 4 kg for the placebo group, 2.47 kg for the 0.3 mg once daily varenicline group, 2.14 kg for the 1 mg once daily group, 1.96 kg for the 1 mg twice daily group and 1.68 kg for the bupropion group.

2. Behavioral Intervention [38]

481 participants were recruited in Switzerland between June 2002 and January 2006 in a two-arm, randomized controlled trial to quit smoking. The two arms are the physical activity group and the control group. Participants in both groups attended a 9-week program with weekly sessions that included smoking cessation intervention with nicotine replacement products and counselling. In addition to the smoking cessation intervention, the physical activity group followed a 9-week physical activity intervention with structured and lifestyle components. Three follow-up visits were conducted at week 10, 26 and 52 after the beginning of the smoking cessation intervention.

There was a total of 229 individuals in the physical activity group and 252 in the control group. 127 and 155 were successfully followed-up at 1 year for the physical activity and control group, respectively. At week 52, the weight gain was 3.1 kg (sd = 0.5) in the physical activity group and 3.7 kg (sd = 0.3) for the control group from baseline. For carbon monoxide (CO) verified sustained quitters at week 52, the average weight gain was 4.4 kg (sd = 0.9) in the physical activity group and 6.2 kg (sd = 0.5) in the control group, with a marginally significant different ($p = 0.06$).

3. Electronic cigarettes [33]

Authors conducted a medical records review of patients with cardiorespiratory conditions regularly followed-up at the outpatient clinics of four Italian hospitals, over a period of around 3.5 years (March 2012 to December 2015). Electronic cigarette users reported at least use on two consecutive follow-up visits. Data from three clinic visits were collected and analyzed. Follow-up visit 1 and follow-up visit 2 were carried out at 6 months and 12 months after baseline visits, respectively.

This study included a total of 223 subjects. These individuals were divided into four major groups: 1. EC users (exclusive users and dual users); 2. Regular smokers (non-EC); 3. Quitters: subjects who reported sustained smoking abstinence after completing a cessation program; 4. Exclusive EC users. Exclusive EC users are those who do not smoke cigarettes while dual users are those who report EC use in combination with cigarette smoking. Dual usage was reported by approximately 50% of EC users in group 1. Quitters experienced on average 4.5% weight gain from baseline in 12 months while EC users (including dual users) put on only 1.5% of baseline weight. At 12 months, the weight change between exclusive EC users and dual users are not statistically different.

Appendix 3.D Model outcomes in one-year increments in Scenario I.

Figure 3.D.1 Average cumulative survival probability under the NHIS sample in Scenario I, status quo vs electronic cigarette.

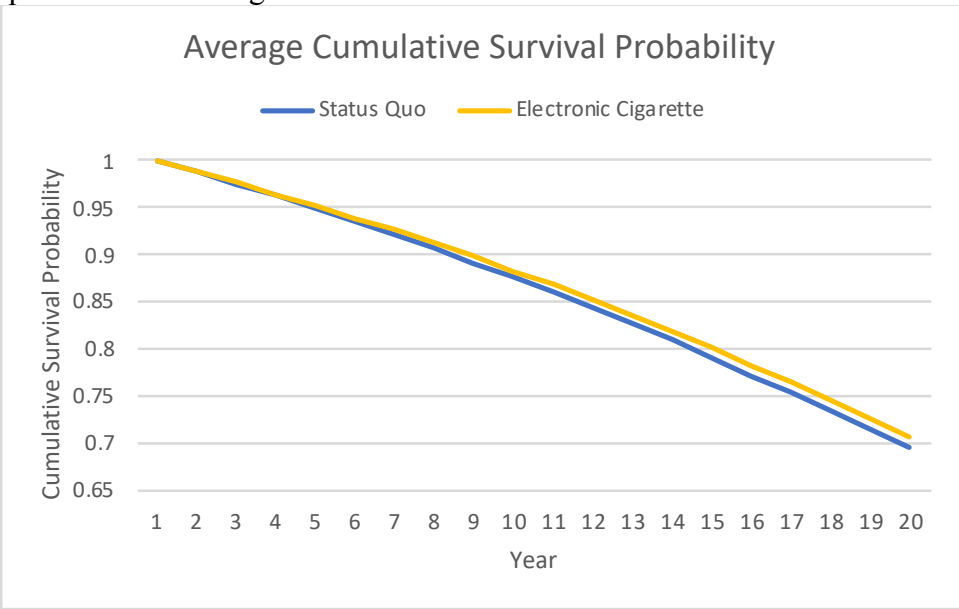


Figure 3.D.2 Average cumulative survival probability under the NHIS sample in Scenario I, status quo vs physical activity.

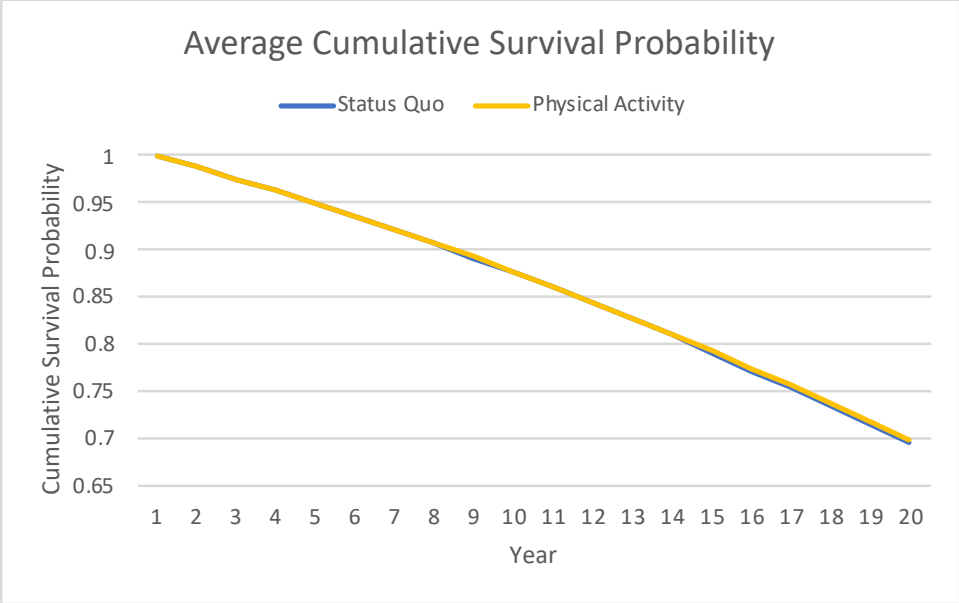


Figure 3.D.3 Average obesity prevalence under the NHIS sample in Scenario I, status quo vs electronic cigarette.

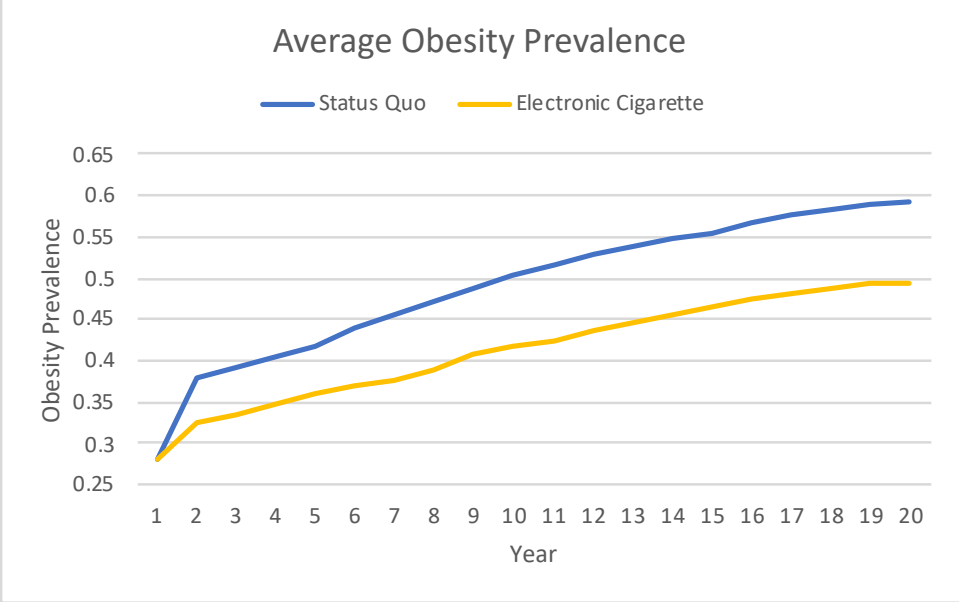
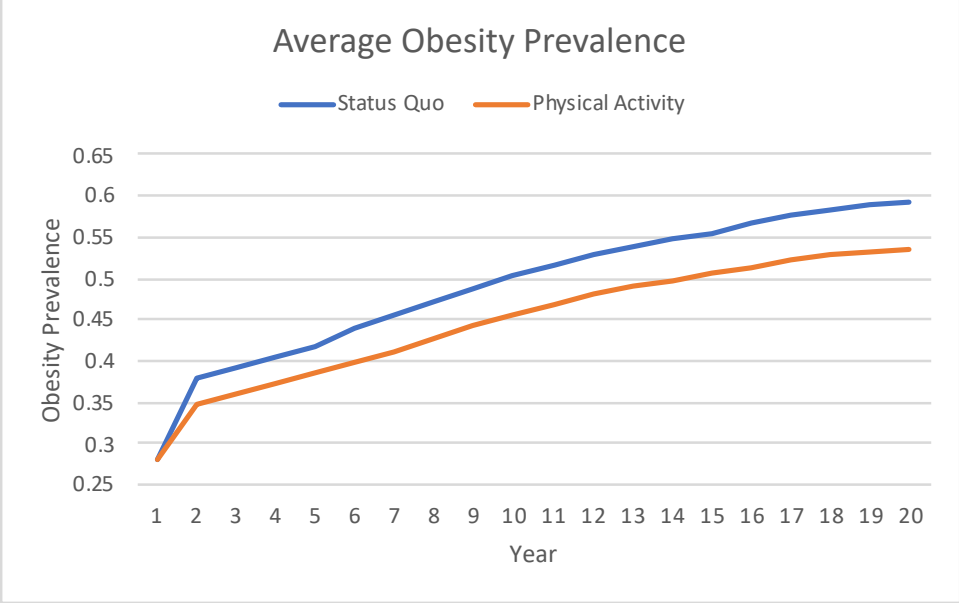


Figure 3.D.4 Average obesity prevalence under the NHIS sample in Scenario I, status quo vs physical activity.



Appendix 3.E Model outcomes in one-year increments in Scenario II.

Figure 3.E.1 Average cumulative survival probability under the random sample in Scenario II, status quo vs pharmacotherapy.

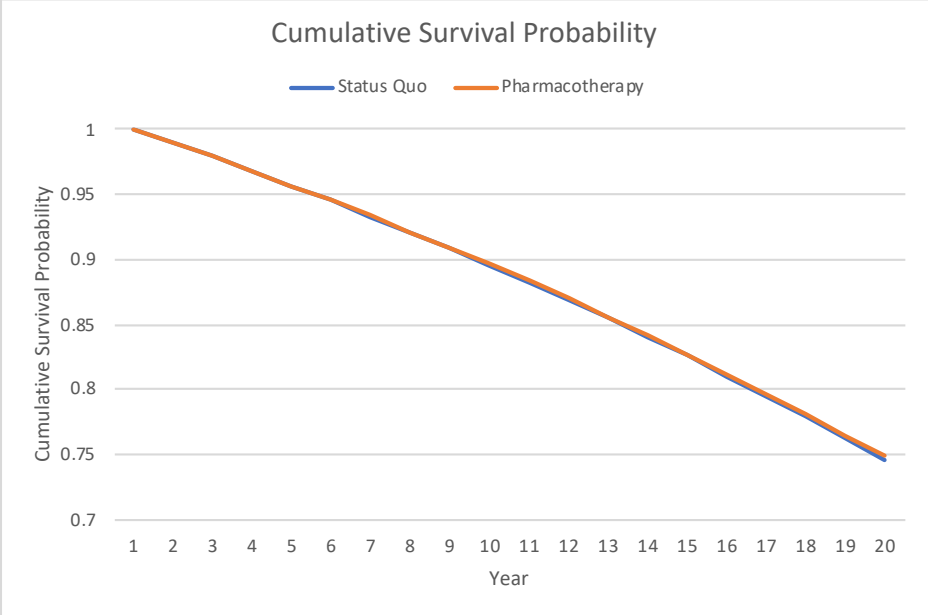


Figure 3.E.2 Average cumulative survival probability under the random sample in Scenario II, status quo vs electronic cigarette.

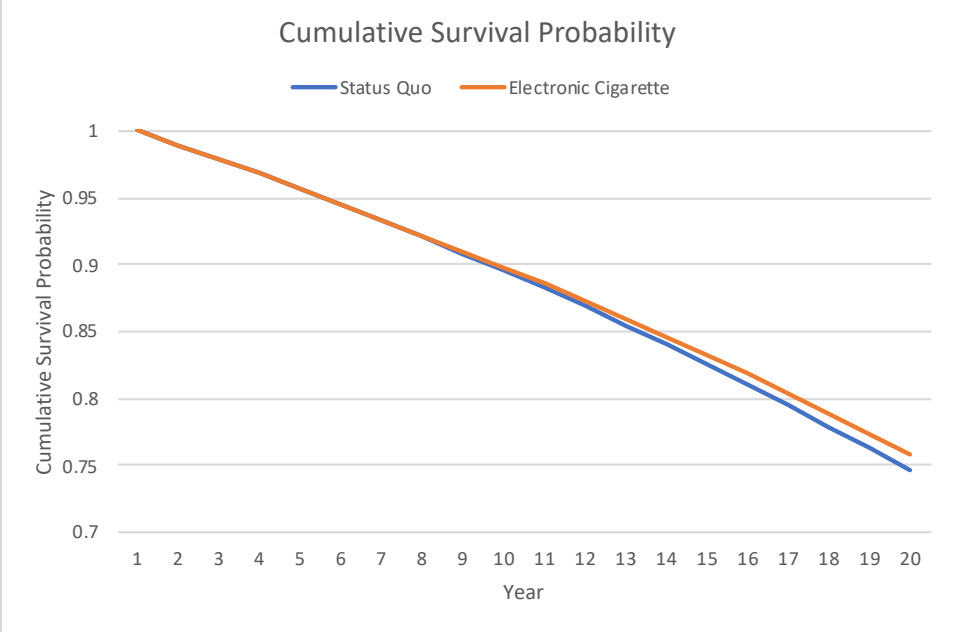


Figure 3.E.3 Average cumulative survival probability under the random sample in Scenario II, status quo vs physical activity.

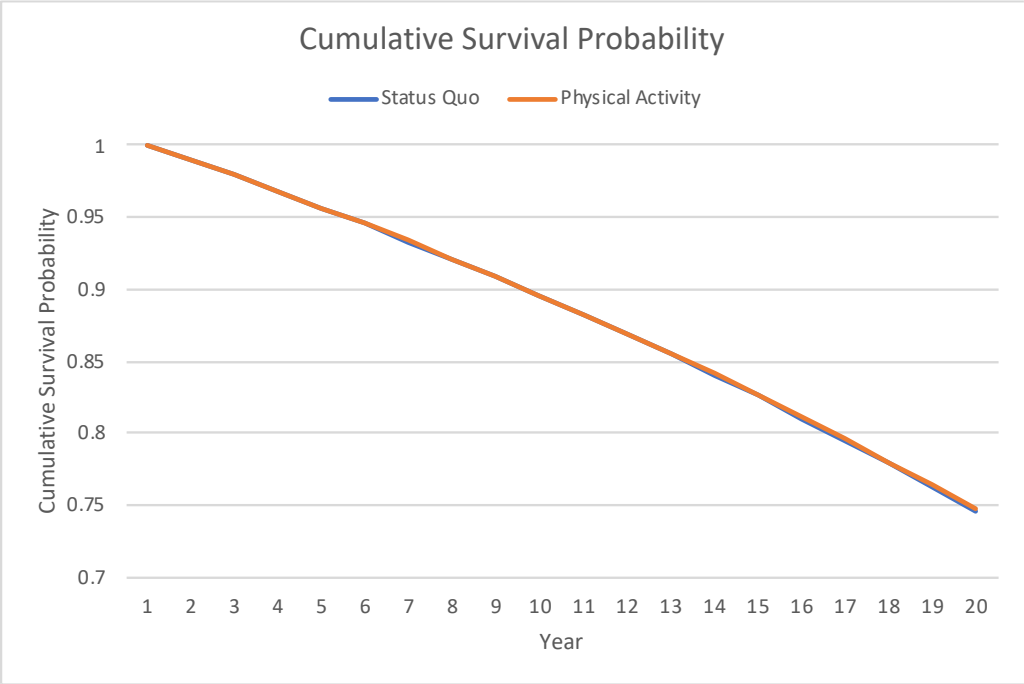


Figure 3.E.4 Average obesity prevalence under the random sample in Scenario II, status quo vs pharmacotherapy.

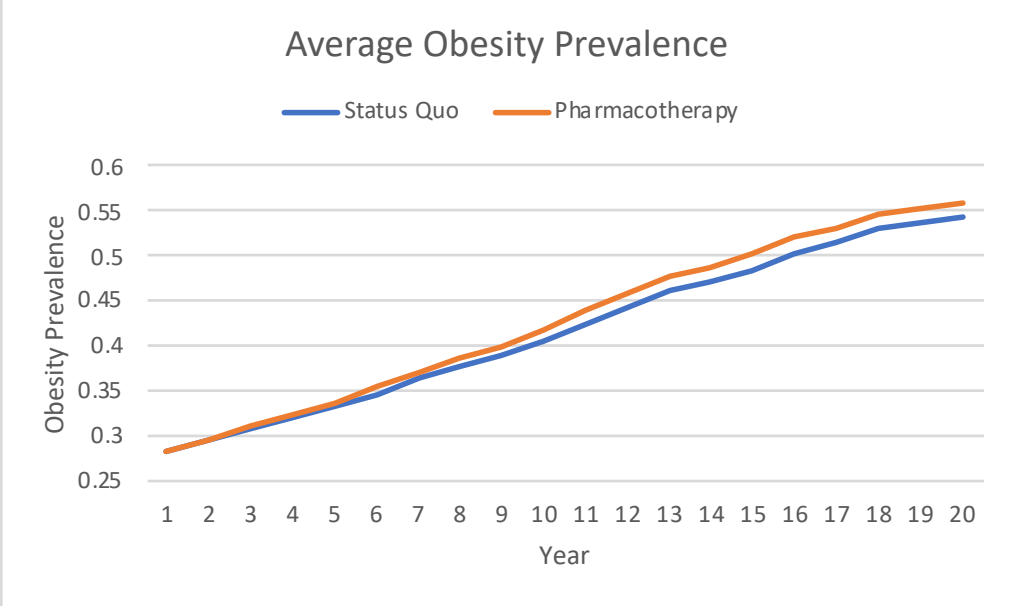


Figure 3.E.5 Average obesity prevalence under the random sample in Scenario II, status quo vs electronic cigarette.

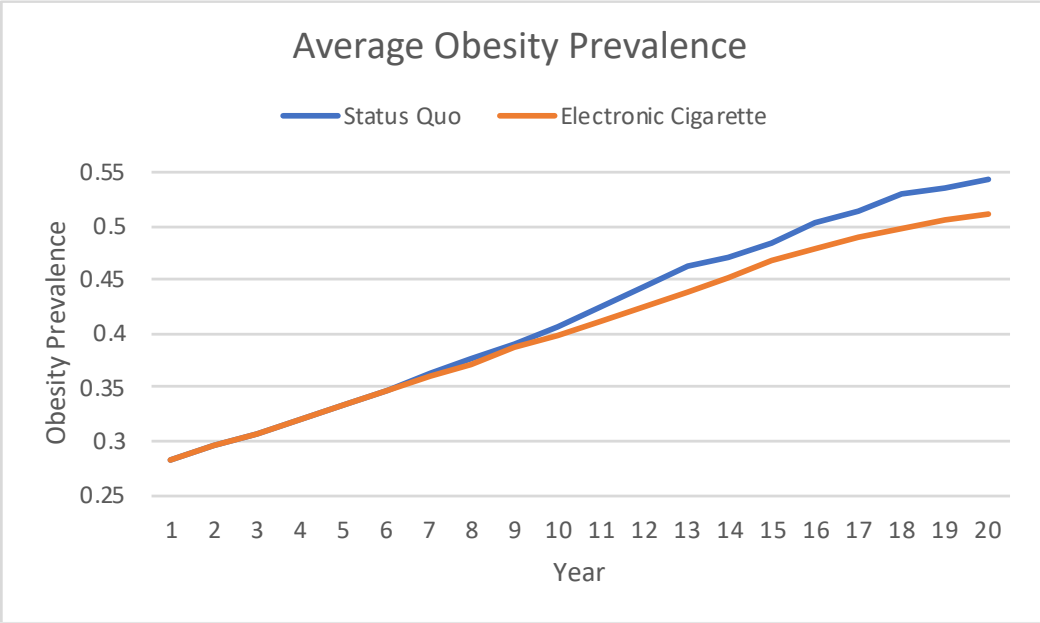


Figure 3.E.6 Average obesity prevalence under the random sample in Scenario II, status quo vs physical activity.

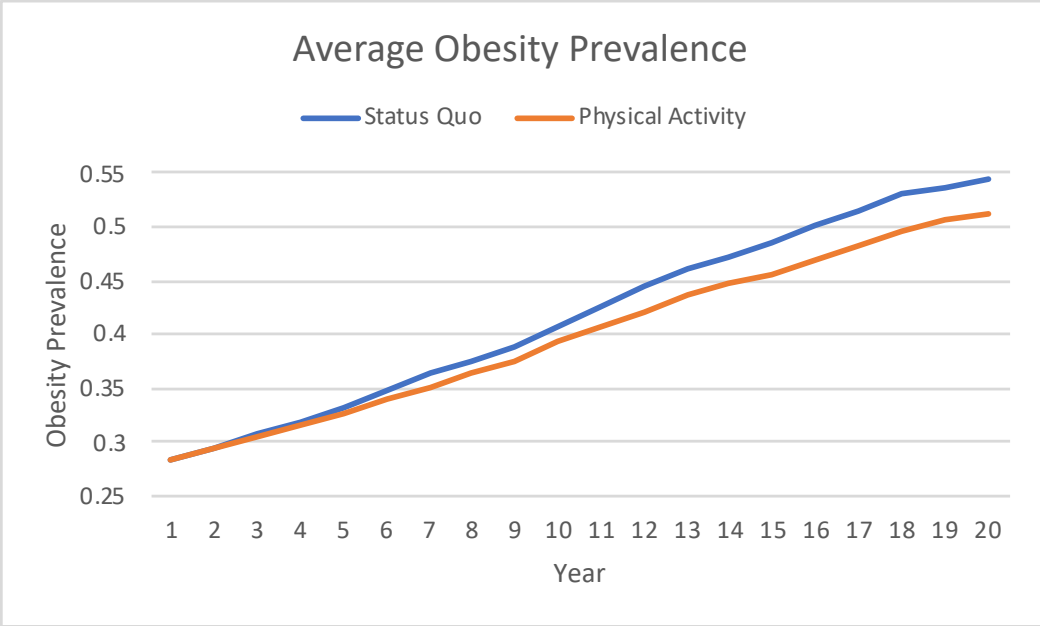


Figure 3.E.7 Average cumulative survival probability under the NHIS sample in Scenario II, status quo vs pharmacotherapy.

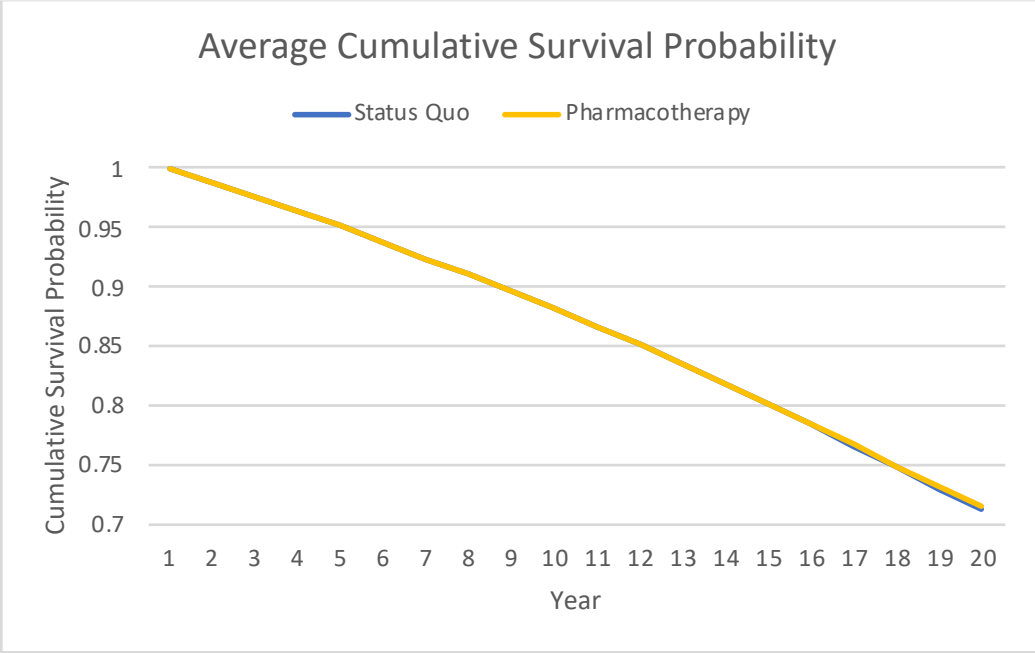


Figure 3.E.8 Average cumulative survival probability under the NHIS sample in Scenario II, status quo vs electronic cigarette.

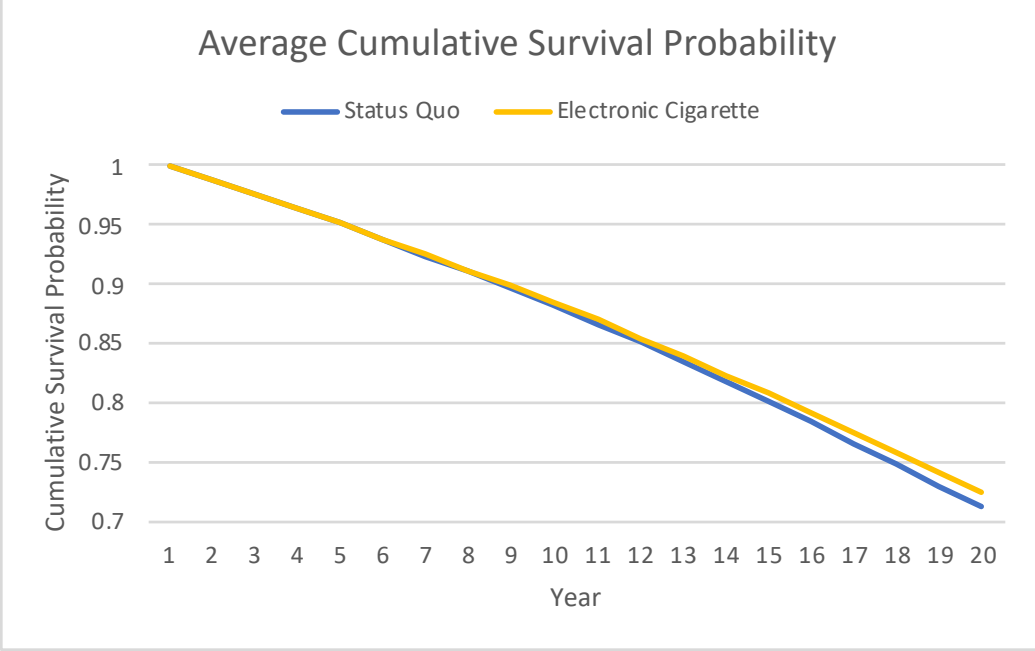


Figure 3.E.9 Average cumulative survival probability under the NHIS sample in Scenario II, status quo vs physical activity.

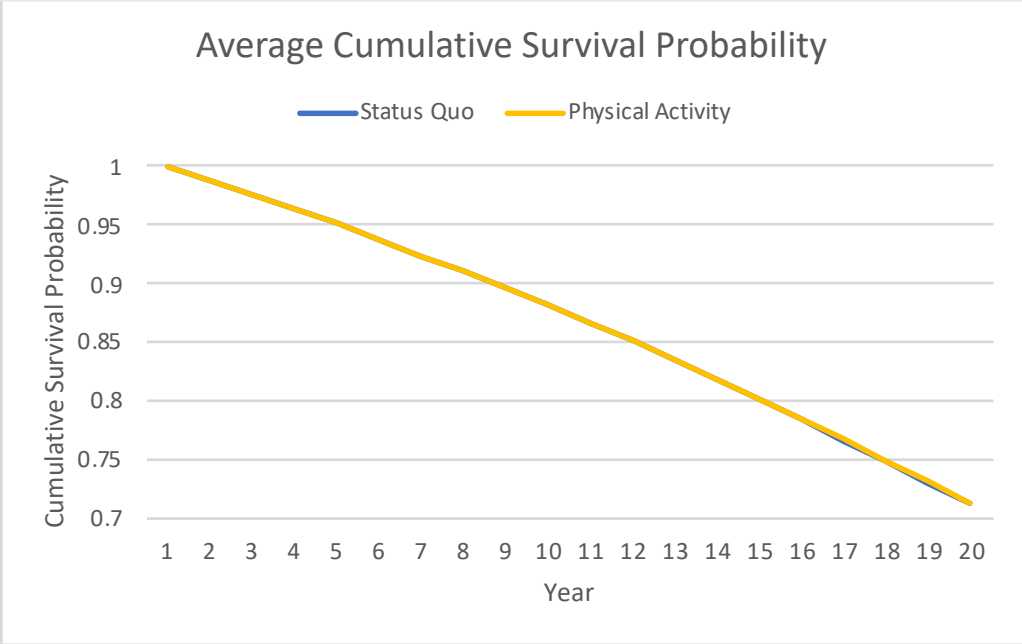


Figure 3.E.10 Average obesity prevalence under the NHIS sample in Scenario II, status quo vs pharmacotherapy.

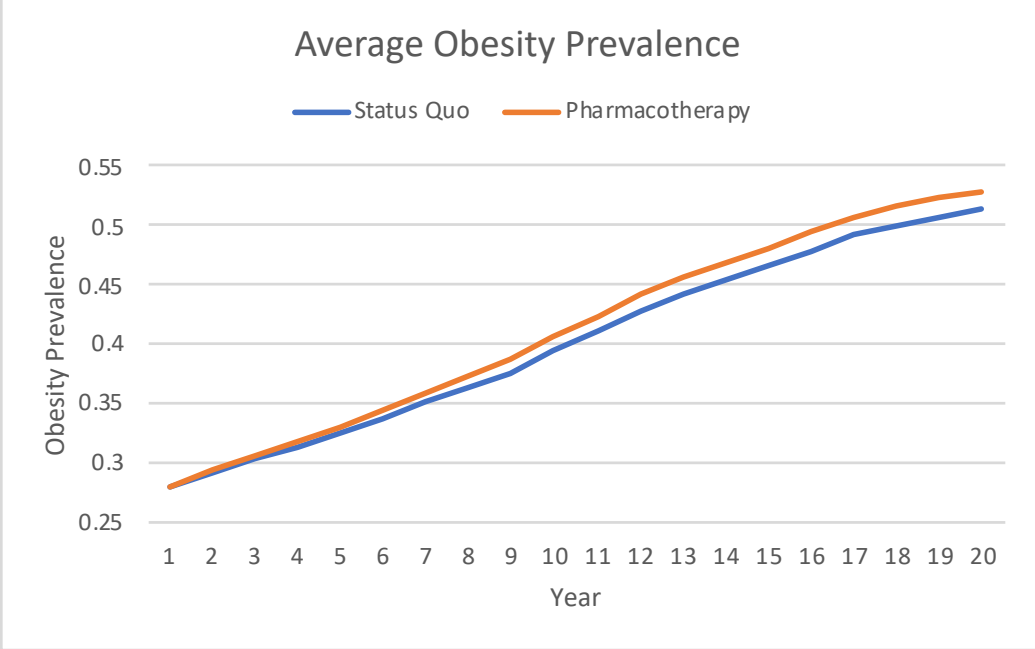


Figure 3.E.11 Average obesity prevalence under the NHIS sample in Scenario II, status quo vs electronic cigarette.

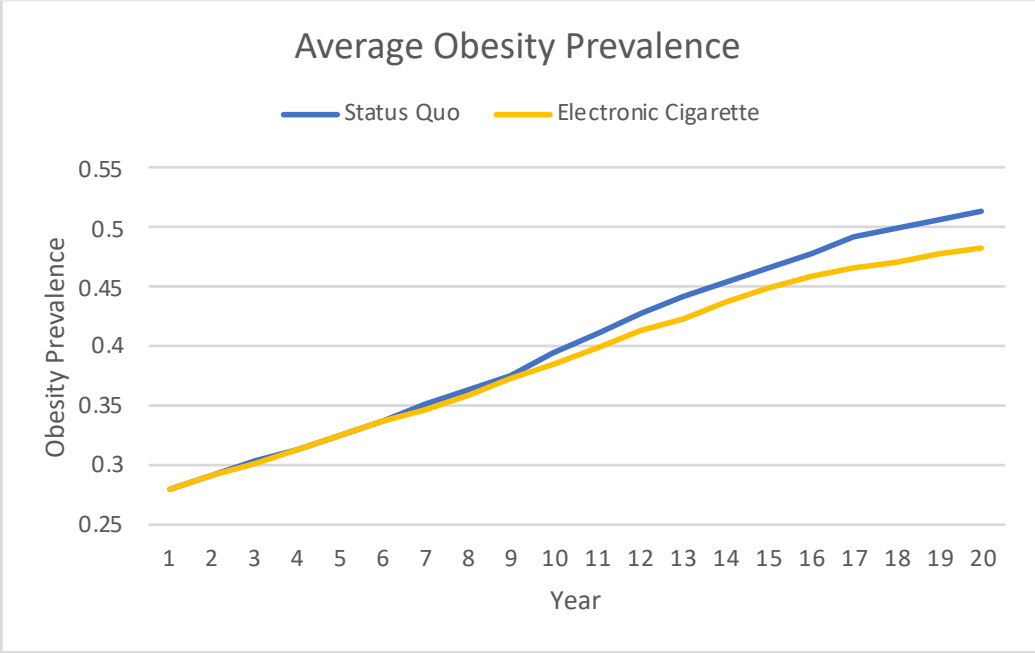


Figure 3.E.12 Average obesity prevalence under the NHIS sample in Scenario II, status quo vs physical activity.